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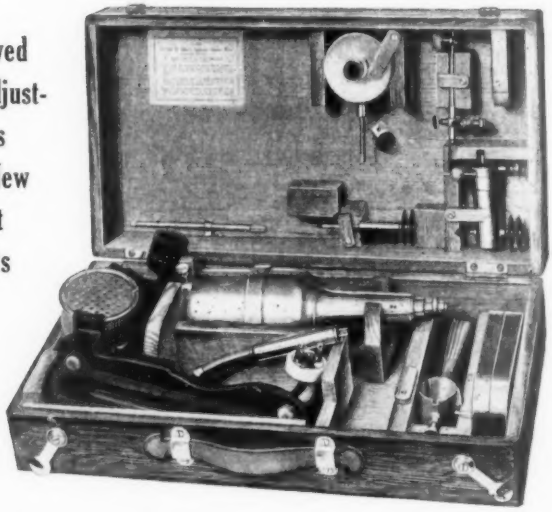

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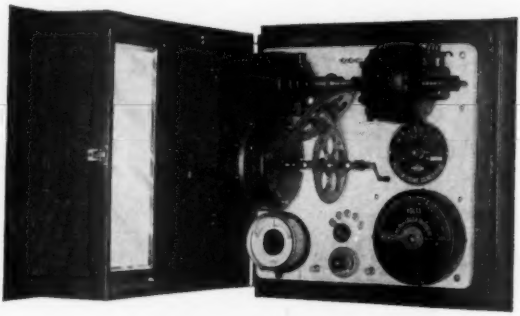

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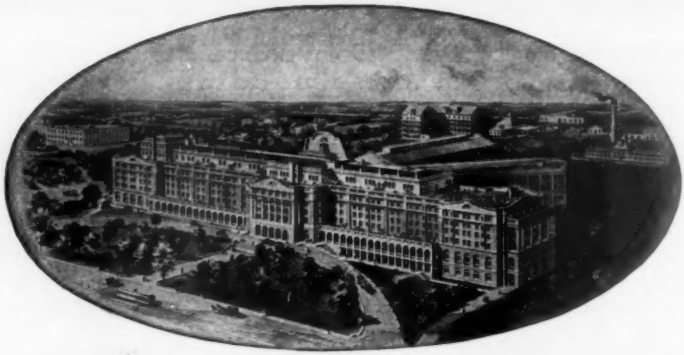

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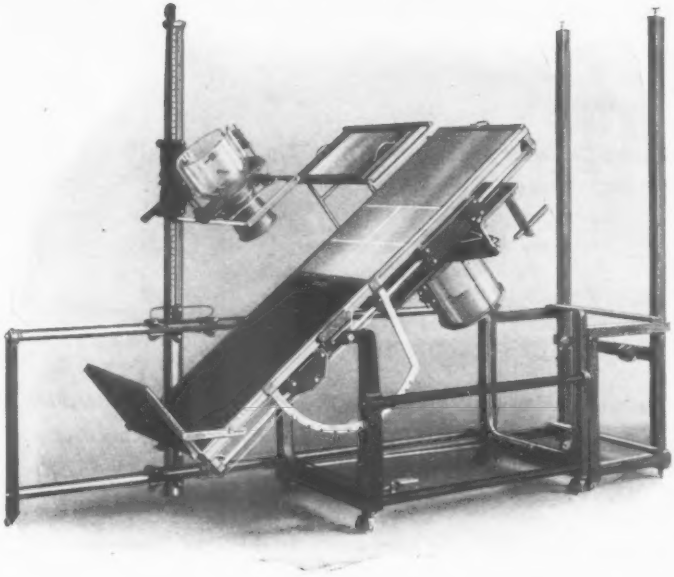

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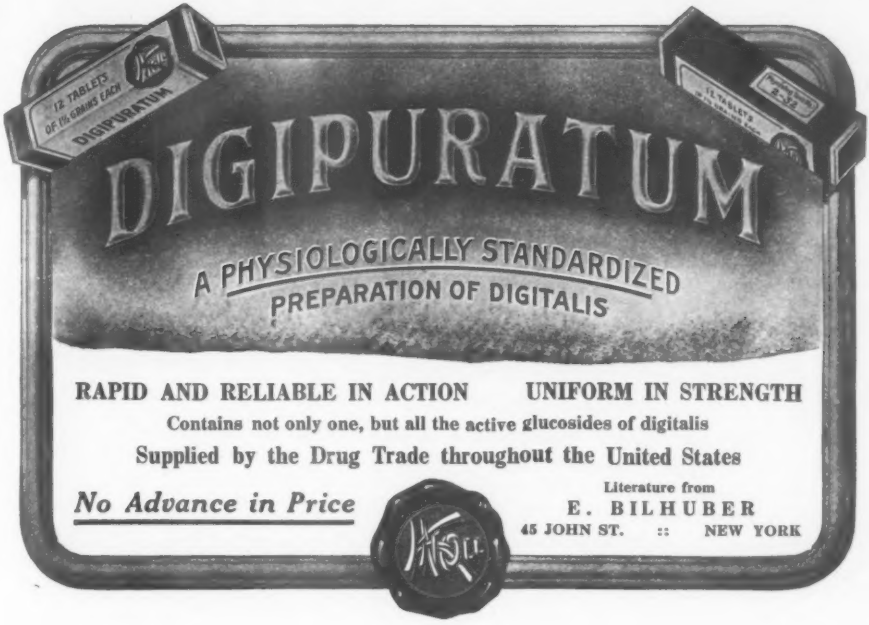
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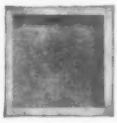

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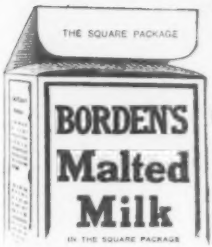

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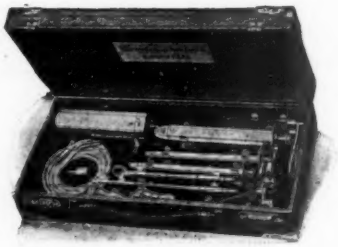

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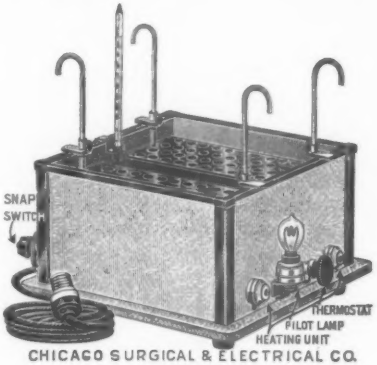

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LIEUTENANT-COLONEL JOHN McCRAE

JOHN McCRAE, B.A., M.D., M.R.C.P. (LONDON),
LIEUTENANT-COLONEL, C. A. M. C.

IN FLANDERS FIELDS.

In Flanders fields the poppies blow
Between the crosses, row on row,
That mark our place; and in the sky
The larks, still bravely singing, fly
Scarce heard amid the guns below.

We are the Dead. Short days ago
We lived, felt dawn, saw sunset glow,
Loved and were loved, and now we lie
In Flanders Fields.

Take up our quarrel with the foe:
To you from failing hands we throw
The torch; be yours to hold it high.
If ye break faith with us who die
We shall not sleep, though poppies grow
In Flanders Fields.

(LONDON "PUNCH," 1915.)

JOHN MCCRAE, physician, soldier and poet, died in France from pneumonia, complicated by meningitis, on January 28, 1918. He was born in Guelph, Ontario, in 1872, the son of Colonel and Mrs. David McCrae, who survive him. His primary education was received in the Guelph schools, and later he entered the University of Toronto, graduating in arts in 1894 and in medicine in 1898, being the gold medallist of his year. He was an intern in the Toronto General Hospital, the Garrett Hospital at Mt. Airy, Md., and the Johns Hopkins Hospital, and then accepted a Fellowship in Pathology at McGill University under Professor J. G. Adami, to whom he became most intimately attached. In 1904 he received the conjoint degrees of M.R.C.S. and L.R.C.P., and later took the M.R.C.P. (London). He had lived in Montreal since 1900, and his work in medicine was chiefly identified with the Medical School of McGill University and the Royal Victoria Hospital. For several years he served as pathologist at the Montreal General Hospital, primarily under the guidance of Wyatt Johnston, and after his

death was in charge of the department. For a number of years he was professor of pathology at the University of Vermont. In 1905 he was elected associate physician to the Royal Victoria Hospital and physician to the Alexandria Hospital (for infectious diseases) in Montreal. He continued his association with the department of pathology, and until his death held the dual position of lecturer in pathology and lecturer in medicine at the Medical School, McGill University.

John McCrae was a born teacher. He loved the simple exposition of the pathology of disease and avoided at all times beclouding the subject with the abstruse and intricate. His demonstrations were impressive and sharp was his criticism, each point being made with a snap which reminded one that he was a student of Carlyle. If contrast was required in teaching it was used even to the grotesque. To the student it was a new departure from the uninspiring lecture, and he revelled in it. I have repeatedly met students who had sat under him and they never stinted their praise of his teaching. His simple methods demanded a firm rule and his class-room reflected a stern but congenial relation of master and student. The ward teaching was his particular delight, and when didactic lectures fell to his lot he converted them as nearly as possible into practical demonstrations by bringing as much pathological material to the lecture room as was possible. The students loved him for the interest he always displayed in their difficulties and because he showed the human side in medicine. They learned from him the unselfish duty of the physician to human distress in all walks of life. They loved him, too, because he never feared to step from the dignity of the teacher to the level of the student.

In 1899–1900 he served with the artillery in South Africa, saw much active service and was in many engagements. Although serving as an artillery officer, he often acted as a medical officer when need required. His military activities brought him the Queen's Medal, with three clasps, and later he was appointed commanding officer of his battery. McCrae had much of the soldier in his make-up. His carriage, his approach and his appearance before an audience marked him of soldierly character. His method of dealing with others was always with that straightforward, square front which we are pleased to recognize as the attribute of a true soldier. He lent no patience to trivial quarrels, and often he would appear as the peacemaker, particularly amid the factions of a junior

faculty who everywhere are Quixotic in their grievances. On the other hand, he would have nothing with the peace-at-any-price party when a quarrel founded upon rights and principles was at stake. He was a man of few words but of decided action. Of his South African experiences he rarely spoke, but we have a number of poems, which though telling us little of the actual warfare, spoke volumes of the thoughts of the man. In 1910 he was invited to accompany Earl Grey, then Governor-General of Canada, on his trip through western Canada, across Hudson Bay and return by way of Labrador. The members of this party never failed to refer to the interest and pleasure which John McCrae infused into the trip. Earl Grey said that "they had travelled 3000 miles and McCrae had a story for each mile."

At the outbreak of war in 1914 McCrae had just arrived in London. He cabled to Canada offering his services and was appointed surgeon to the First Brigade of Canadian Artillery. He was with the guns along the Ypres sector for a continuous period of fourteen months, and was in the thick of the engagements where the Canadian forces made an undying name for their valor. His brigade was behind the area where the first gas attack was delivered and his description of their moving up to hold the front line was most graphic. They were under intense fire for seventeen days, and on one occasion a shell came through his dressing station, but he escaped injury. When in the service of his country he was a man of few words, and few there are who have an appreciation of what he endured. His health was undermined by the strain of constant duty and the conditions under which the men were living in the early period of the war. He did not realize this: in fact, he was reproving himself for the lack of greater sacrifices which he deemed it his duty to make. It was only through the strong appeal of his friends that he consented to accept the post of internist at a base hospital (McGill Unit).

In his medical duties he was equally severe with himself. He sacrified his all for the comfort and welfare of the men under his charge; he was keenly desirous that all the invalided should have the last ounce of care provided, to restore them to health as early as possible. He served for over two years as chief in medicine in the McGill Unit. Just before his death he had been appointed consultant to one of the British Army areas, the first officer of the overseas forces to be so honored. News of this came on the day on

which he was stricken with pneumonia. The attack at first seemed mild, but meningitis developed on the third day and death came two days later.

In medicine John McCrea will be known to us for his admirable teaching, and a number of researches of which his investigations on the relation of extensive superficial burns to duodenal ulcers, acute mycotic aneurysm of the aorta, necroses of the liver and a clinical study of scarlet fever are among the most important. He was co-editor with Professor Adami in the writing of a *Text-book of Pathology*. He edited the department of Pathology and Bacteriology in this JOURNAL for a number of years. However, he is much more widely known to a general audience for his literary essays and poems. During the present war the poems given herewith are among his best. "In Flanders Fields" has received favorable criticism everywhere, and has been republished the world over.

John McCrae leaves a host of friends to mourn his untimely end. All will sorely miss him and his genial presence. Those who knew him will carry forward his torch, not forgetful of the living spirit of the man and a character as true as steel. His last poem, "The Anxious Dead," may almost be regarded as an answer to "In Flanders Fields."

THE ANXIOUS DEAD.

O guns, fall silent till the dead men hear
  Above their heads the legions pressing on
(These fought their fight in time of bitter fear
  And died not knowing how the day had gone).

O, flashing muzzles, pause, and let them see
  The coming dawn that streaks the day afar:
Then let your mighty chorus witness be
  To them, and Caesar, that we still make war.

Tell them, O guns, that we have heard their call,
  That we have sworn, and will not turn aside,
That we will onward, till we win or fall,
  That we will keep the faith for which they died.

Bid them be patient, and some day, anon,
  They shall feel earth enwrapt in silence deep,
Shall greet, in wonderment, the quiet dawn,
  And in content may turn them to their sleep.

(LONDON "SPECTATOR," 1917.)

OSKAR KLOTZ.

THE

AMERICAN JOURNAL

OF THE MEDICAL SCIENCES

APRIL, 1918

ORIGINAL ARTICLES

SPINA BIFIDA OCCULTA:*

(1) WITH EXTERNAL SIGNS, WITH SYMPTOMS.
(2) WITH EXTERNAL SIGNS, WITHOUT SYMPTOMS.
(3) WITHOUT EXTERNAL SIGNS, WITH SYMPTOMS.
(4) WITHOUT EXTERNAL SIGNS, WITHOUT SYMPTOMS.

By Walter M. Brickner, B.S., M.D., F.A.C.S.,

ASSOCIATE SURGEON, MT. SINAI HOSPITAL; ATTENDING SURGEON, BROAD STREET HOSPITAL, NEW YORK; MAJOR, MEDICAL RESERVE CORPS, U. S. A.

By spina bifida one ordinarily means, and the dictionaries so define it, a cleft or deficiency in the bony column with a lesion of the spinal cord or its membranes—usually a protrusion (meningocele, myelocele, or meningomyelocele). This is, I think, a wrong use of the words. Spina bifida means bifid spine, and that individual has a spina bifida who has a cleft in the spine, whether or not there is protrusion of the spinal cord structures. In 1875, when Virchow[1] reported what was supposed to be the first case observed, he coined the term "spina bifida occulta" to indicate a type of spina bifida in which the lesion was concealed beneath the skin. In a search of the literature as far back as 1825 I found, however, two earlier cases[2] that belonged to this type of spina bifida,† and up to 1910 I found recorded about 85 cases. In these the lesion was indicated

* Read, by invitation, before the New Hampshire Medical Society, May 16, 1917.

† The satyrs of the ancients may well have been suggested by instances of spina bifida occulta with short sacral "tails" and club-feet. Concerning satyrs, see Pauly, *Realencyklopädie der Klassichen Alterthumswissenschaft.*

externally most often by a distinct hypertrichosis over the cleft, somewhat less often by a congenital lipoma symmetrically situated over the cleft, occasionally by a nevus, telangiectasis, or scar, or sometimes by a combination of these. Such a spina bifida occulta is most often found in the lumbar or lumbosacral region, but it may occur in the dorsal or even the cervical region. It usually involves only a few vertebral arches, but it may extend through the greater part of the spinal column. The "woman with the horse's mane,"[3] exhibited many years ago in the "dime museums," with a luxuriant mane of hair over the midline of her back, was an instance of such an extensive bifidity. Mere hairiness of the lumbosacral region, which is noted in many individuals, is not indicative of a spina bifida. Such a well-marked hypertrichosis with long hairs as is shown in Fig. 19, is, however, a pretty safe indication that a spina bifida occulta is present, and probably most of the cases of sacral hair growth studied by anthropologists some years ago as an atavism, or a stigma of degeneracy, are instances of this congenital deformity, as first suggested by Recklinghausen.[4] I have found some very interesting records of such lumbosacral hair growth in several members of a family, however, and spina bifida occulta is not an hereditary phenomenon. The hypertrichosis may not appear, or develop prominence, until puberty.

The congenital lipoma associated with spina bifida occulta is not freely movable, but is somewhat attached to the underlying aponeurosis, and is usually quite circular in outline (Fig. 2). In 4 cases operated upon I have confirmed the interesting observation recorded by A. Jacobi,[5] many years ago, that congenital lipomata differ from the other varieties in being unencapsulated and very finely lobulated, in which respect they are much like subcutaneous fat. Not every case of congenital supraspinal lipoma has a bifidity associated with it.

In several cases of spina bifida occulta scoliosis or other spinal deformity is also present, and sometimes there are other congenital malformations.

In his recent work on the *Surgical Diseases of the Spinal Cord* Elsberg explains the phenomena of spina bifida occulta in the following manner: At birth the cord extends down to the lower end of the spinal column; as the individual grows the cord recedes, relatively speaking, until its lowest end is at about the first or second lumbar arch; if, therefore, any nerve roots are congenitally herniated through a spinal column dehiscence, these will undergo stretching as the disproportion in length between the bony and medullary columns develops with the growth of the individual. Of course, however, this must not be accepted as the entire explanation for the following reasons:

1. In some cases with symptoms there is no hernia of the spinal cord structures.

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2. In a few recorded cases, symptoms developed as early as the second year after birth.

3. In some cases, as in one here recorded (Helen H.), the lesion is a teratoma (fibromyolipoma) of the cauda equina.

4. In some cases there is an exostosis in the canal compromising the cord tissues.

5. In many cases there is ample clinical evidence of a lesion in the cord itself.

6. At autopsy in some of the recorded cases dilatation of the spinal canal or degeneration of various tracts has been demonstrated.

At operations and autopsies these abnormalities have been found: A cleft of varying length or breadth in one or more arches accompanied by one of the following conditions: (1) The cleft may give passage to a distinct meningocele; (2) the cleft is closed by a tough membrane adherent to the overlying skin or non-encapsulated fat and connective tissue; (3) the membrane is perforated by a dense band attached to the subcutaneous tissues without and compressing the cord structures within; (4) lipomatous tissue within the canal is concealed by this membrane; (5) the cleft discloses the bulging dura mater; (6) an exostosis within the canal compromises the cord tissues; (7) a myofibrolipoma extends through the cleft into the bony canal distorting and compressing the cord and its nerve roots; (8) dilatation of the medullary canal; (9) degeneration of cord tracts.

The symptoms arising from spina bifida occulta usually appear during adolescence or early adult life, but not rarely they develop during childhood, and occasionally they first appear during middle life. These symptoms may be one or more of the following: Incontinence of bladder or rectum; sensory paralyses; motor paralyses; disturbances of the reflexes; trophic ulcerations and gangrene. Symptomatically, therefore, spina bifida and spina bifida occulta are one. It must be noted, too, that pathologically spina bifida and spina bifida occulta do not greatly differ. Tumor tissue sometimes accompanies evident spina bifida; hypertrichosis sometimes does; an evident meningocele is occasionally partially concealed by a congenital lipoma; and, finally, there are reasons for believing that a spina bifida sometimes shrinks, leaving only scar tissue or other index to a spina bifida occulta.

Of the type of spina bifida occulta with symptoms and with external signs, by which I mean lipoma, hypertrichosis or nevus, the following 3 cases are interesting examples.

GROUP I.—*Spina Bifida Occulta, with External Signs, with Symptoms.*

Sigrid H., female, aged eighteen years, came under my care in December, 1907, for an extensive ulceration of the inner and flexor aspects of the great toe of the left foot, indolent in appearance, and with foul discharge.

She was a native of Jënköping, Sweden, where, at the age of seven, the second left toe was ulcerated from what was presumed to be frost-bite. This ulceration recurred each winter and, at the age of twelve, partial amputation of the toe was required.

Three years later (aged fifteen) the stump of the toe ulcerated and the phalanges necrosed. Six months later the fifth toe ulcerated, refused to heal, and was amputated.

She never had vesical, intestinal, or motor disturbances. She never noticed anesthesia in either foot or leg. No lightning pains, but these ulcerations were mildly painful. At the appearance of each fresh ulceration she said she had malaise, headache, nausea, and fever, and the toe would first appear red.

Observations of this patient and the next one to be reported confirmed this statement: preceding and accompanying each fresh ulceration, both patients felt ill and had a rise of temperature to 103° or 104°. The appearance of a fresh ulceration could thus be accurately predicted.

The girl was referred to my surgical from the neurological department of the Mount Sinai Hospital Dispensary as a purely surgical case, and with the assurance that there was no neurological lesion. I mention this not in criticism, but to illustrate how even a trained neurologist may overlook a spina bifida occulta until a single experience teaches him to be on the lookout for it.

The foul ulceration for which the patient came to me has been briefly described. The first phalanx was necrotic. The wound was laid open and this bone was removed. Slowly the wound healed, *without granulating*, after the head of the first metatarsal spontaneously discharged. Specific medication seemed to exercise no influence. The dorsalis pedis, popliteal and posterior tibial vessels pulsated normally. The urine was negative. The foot condition and the patient's nativity suggested the possibility of leprosy.

Dr. Prince A. Morrow, who was kind enough to examine the patient for me, wrote: "I think the diagnosis lies between syringomyelia and leprosy, probably the latter. I have seen quite a number of unilateral manifestations due to leprosy, although the dystrophic changes are usually symmetrical. The rather characteristic plantar ulcers and the anesthetic ulcers are in favor of leprosy."

In April, 1908, the two remaining toes (third and fourth) ulcerated and the two last phalanges of these were removed, leaving stumps. The foot was then quite edematous but not painful, although there developed at this time a plantar ulcer near the base of the third toe (Fig. 1). Smears from the ulcers showed no lepra bacilli.

I had the patient admitted to the second surgical division of the Hospital (Dr. Lilienthal) for further study. I abstract from her history the following positive findings:

Over the lower lumbar and upper sacral region extending down

from the third lumbar vertebra is a circular tumor about four and a half inches in diameter symmetrically situated over the midline

FIG. 1.—Sigrid H., September, 1908. Plantar ulcers at base of third toe and over the head of the second metatarsal. Note stumps of toes.

FIG. 2.—Sigrid H. Congenital lipoma over spina bifida occulta.

(Fig. 2). It is about one and a half inches thick in its center, of doughy consistency, painless and not tender, moderately movable over the deep structures and adherent to the skin at its center. This

tumor was present since birth and has grown with the patient. In the spine no cleft can be felt. No hypertrichosis. The left foot is as above illustrated and described. Blackish, rugous epithelium covers part of the third toe where it abuts against the great toe. The plantar ulcer is small, deep, conical, lined with granulations, but does not extend to the bone. Reflexes: abdominal—right, active; left, absent: gluteal—absent on both sides: knee-jerks—right, feeble; left, absent: achilles—right, feeble; left, absent. No Babinski or

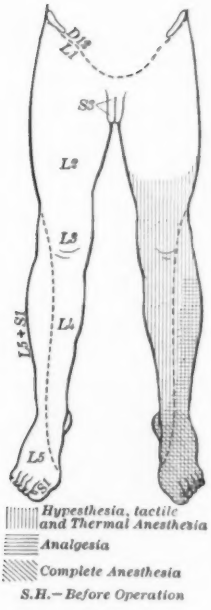

FIG. 3

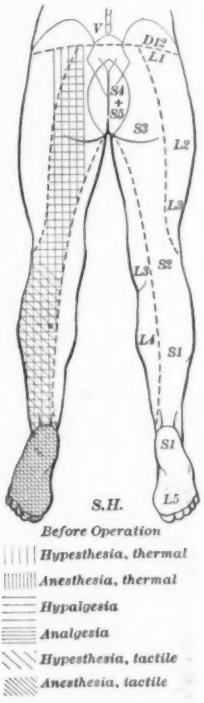

FIG. 4

Oppenheim phenomenon. Motor power of lower extremities normal and equal. Gait normal. No ataxia. Tests show sensory paralyses of the left lower extremity as illustrated in Figs. 3 and 4. Roentgenogram shows a cleft in the *left* lamina of the fourth lumbar arch. The fifth lumbar arch is not of normal shape (Fig. 5). Clinical diagnosis: spina bifida occulta associated with congenital lipoma over the lower end of the spine; cleft in the fourth lumbar arch with a lesion involving the posterior fourth and fifth lumbar and first, second, and third sacral roots or cord segments.

Fig. 6, taken from Sobotta's *Anatomy*, illustrates how pressure on the cauda equina at the level of the fourth lumbar vertebral arch would affect the posterior roots below the level of the third

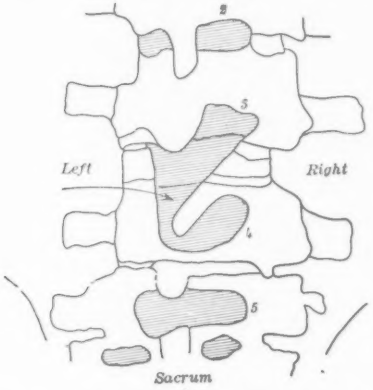

Fig. 5.—Sigrid H. Tracing of roentgenogram of lateral spina bifida.

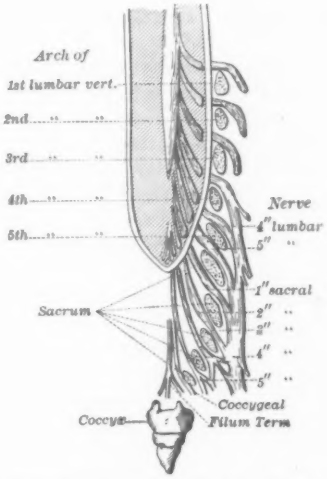

Fig. 6.—Emergence of the posterior nerve roots between the bony arches. Illustrating that pressure on the cauda equina at the level of the L4 arch would affect these roots below the L3 root. (After Sobotta.)

lumbar root, and how, if sufficiently circumscribed, some of the lumbar and sacral roots on one side only might be involved. This was, in fact, just the condition found at

Operation (Brickner and Elsberg), July 3, 1908. Curvilinear incision over the upper border of the tumor, which was found to be a large collection of unencapsulated fat arranged in very small lobules like subcutaneous fat and practically free from bands of fibrous tissue. Incision was carried through this down to the lumbar aponeurosis and the spinous processes. These were found normally disposed, but just to the left of the fourth lumbar spine there was a distinct hiatus in the muscles—an opening lined with fascia which here dipped down toward the spinal canal. This opening easily admitted the finger-tip. Through it issued a strong, apparently dense columnar structure. This structure terminated in the lipoma, but it had no attachment to the skin. With some of the attached fat it was dissected free and dragged through the opening. Incision through the fibrous envelope revealed a serous lining, within which were several nerve fibers. A probe could be passed along these into the spinal canal, and cerebrospinal fluid escaped alongside it. The dural extension thus exposed was further opened, revealing that the hernia was a meningeal sac containing four or five distinct nerve roots of the cauda equina, which bent upon themselves and reëntered the spinal canal. At the point at which these roots were flexed they were found somewhat adherent to the sac, which was at this point much constricted. Distal to this point the meningocele then expanded, but no fluid was noted in this expansion.

The sac was removed, the nerve roots were reduced into the spinal canal, and the dura was sutured over them. The lumbar aponeurosis was closed over the hiatus by a plastic operation.

Following the operation the patient complained of numbness in both lower extremities for a few days, but there was no other untoward effect, and the wound healed *per primam*. Soon after the operation the patient volunteered the statement that her left lower extremity felt more normal, more like the right, than it had for many years before, and improvement was noted in the sensory paralyses, as indicated in Figs. 7 and 8.

Pathological examination of the amputated sac and of the adherent fat revealed no features of special note.

The patient was discharged from the hospital August 4 and readmitted August 25. The ulceration in the third toe had progressed and the plantar ulcer had also increased in size, exposing the head of the second metatarsal bone.

By September 28 these ulcers were healed without any bone necrosis, and the patient was again discharged with a raised sole on her right shoe and crutches.

In November the plantar ulcer reopened, as usual with a rise of temperature to 103°. Surrounding the ulcer there developed a thick, soft callus in spite of the fact that the patient had not put her foot on the ground for many months. During the next few

months the ulcers improved and relapsed and others developed. The patient was readmitted to the hospital.

July 26. Typical Pirogoff amputation (Brickner). All the bloodvessels appeared quite normal. Primary union.

Pathological report of the amputated foot showed—to summarize—practically normal vessels, but marked bone atrophy. This atrophy was found in portions of the bone remote from the inflammatory process. The nerves of the foot showed no degenerative changes. In the immediate vicinity of the ulcers there were moderate inflammatory changes in the nerves.

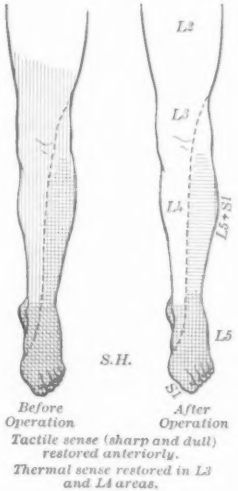

FIG. 7

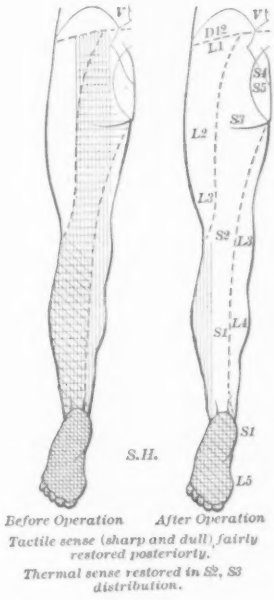

FIG. 8

Contrary to advice the patient insisted upon wearing a shoe containing a cork foot. The pressure of this cork against the anterior aspect of the stump caused an ulceration of the skin within a month after the patient was discharged from the hospital.

Between October, 1909, and May, 1910, the patient neglected treatment, reporting but rarely, the skin necrosis meanwhile extending into a large shallow ulcer surrounded by a dense callus. The raw area was granulating, bled easily, and would probably have healed under good treatment if the patient would have abandoned the shoe, which she insisted upon wearing.

I lost track of her between May, 1910, and January 30, 1911, when she reported again. The ulceration was then much reduced in size, but a gangrene of the heel portion, extending almost to the bone, was present.

February 6, 1911. Osteoplastic amputation through the leg (Brickner). Primary union. Discharged from the hospital.

Soon after that I lost all trace of the patient until in the winter of 1917, through the courtesy of Dr. John Moorhead, I saw her again in the Harlem Hospital. Since my last preceding note she had married and had two healthy children, free from deformity. She had no difficulty in parturition, had no bladder or rectal trouble. During the past year she had lost the fourth and fifth toes of the *right* foot following infection of a corn. She had gone to the Harlem Hospital because of a deep indolent ulceration of the amputation stump (left leg), which had been increasing in size for the preceding six months. During all these six years she had worn an artificial leg pressing against the stump and from time to time had had superficial ulcers, which, however, healed. This deep ulceration did not extend to the bone, and an *x*-ray picture showed that the bone was not at fault.

Sensory examination in March, 1917, showed in the outer half of the stump below the knee, thermal and tactile hypesthesia as previously, but the right foot and the lower portion of the right leg posteriorly now also show thermal and tactile hypesthesia. The large, deep ulcer of the stump gradually healed.

Whether or not the release of the nerve roots in the spinal hernia obviated any other troubles (*e. g.*, vesical) it would be mere speculation to say.

Our experience with this case suggested to Dr. Elsberg the diagnosis in the case of the following patient, whom he had had under observation for several years at Montefiore and Mount Sinai Hospitals:

Helen H., aged twenty-six years, single, hairdresser. No member of the family, direct or collateral, had had a similar trouble. One sister has a congenital club-foot, unilateral; no other congenital deformities in the family.

Patient entirely well until nineteen. Then, in 1901, she noticed two suppurating ulcers, the size of dimes, in the calf of the left leg, which had appeared spontaneously and painlessly and healed painlessly in two weeks. A month later severe shooting pains in the calf of the left leg and especially the left foot. These continued for a few weeks, when ulcers appeared on the fifth toe and on the sole of the left foot, which swelled. These healed and broke open again repeatedly during the next nine months, and the toe was then amputated. Sensory paralysis was then noted in the other toes, and later in the left foot, leg, and thigh. During the next few years she similarly lost the second, third, and fourth toes, and had ulcers

of the sole and heel of the same foot. She had no urinary disturbances, but had had diarrhea for several weeks before admission to the hospital, July 20, 1908.

Physical examination revealed the following positive findings: Spine normal to inspection and normally flexible. Over the upper end of the sacrum and the fifth lumbar vertebra is an almost inconspicuous swelling, due apparently to a small accumulation of fat; it has no distinct boundaries, but extends almost to the sacro-iliac joints. It is asymmetrical in depth, being thickest on the right side. The back is covered with light colored lanugo, but over the left side of this lipoma there is a small tuft of very small, short, black hairs, about a dozen in number, about 1 cm. in length, and very slender. The lumbar spinous processes are easily palpable and appear well formed except the fifth, which feels small and lies a trifle to the right of the line through the others. Just below the fifth lumbar process, viz., at the site of the above-mentioned swelling, the fingers on deep pressure sink into a shallow depression, and bony contact is not transmitted. The rest of the sacrum and the coccyx appear normal to palpation.

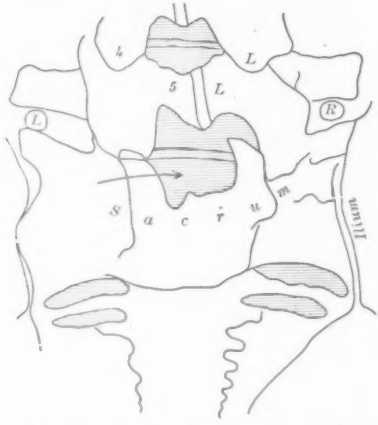

FIG. 9.—Helen H. Tracing of roentgenogram of spina bifida.

There is a sacrococcygeal dimple, but no sinus. Roentgenogram shows the spina bifida illustrated in Fig. 9. It consists of a distinct and extensive hiatus in the first sacral arch, most marked on the left side. The fifth lumbar arch is complete, but it is asymmetrical and narrow, the spine lying to the right of the midline.

The lower extremities show an atrophy of the left calf and exaggeration of the left knee-jerk. No ankle-clonus on either side. The outer side of the single remaining toe of the left foot is practically

denuded of epithelium from a recent bleb. There is a plantar callus over the head of the second metatarsal. Scars of ulcers on the calf and over the tendo Achillis. Locomotion and station normal. Abdominal reflexes lively and equal. The right lower extremity shows no disturbances of sensation. The left lower extremity and left side of the anogenital region show the sensory paralyses illustrated in Figs. 10 and 11, indicating, with the trophic disturbances, an involvement of the fifth lumbar and all the sacral nerve roots or segments on that side and a slight involvement of the second, third, and fourth lumbar roots.

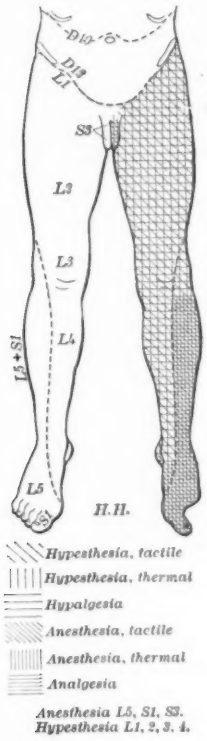

FIG. 10

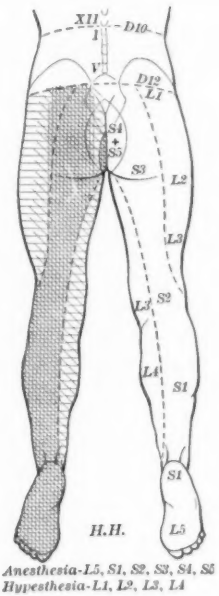

FIG. 11

Operation (Elsberg and Brickner), July 27, 1908. Slightly oblique transverse incision across the upper level of the sacrum carried down through the underlying fat, unencapsulated and finely lobulated, to the lumbar aponeurosis. Over the upper end of the sacrum this aponeurosis was found to dip in somewhat like a shallow funnel, but

there was no opening in it and no structure passed through it. The depressed portion of the aponeurosis was circumcised, and this was dissected up from the underlying muscle, which was retracted, exposing a mass of fat and connective tissue extending down between the muscles in the midline and firmly attached to the under surface of the lumbar aponeurosis. No dura or congenital band was here encountered, but further dissection showed that the mass of fat and connective tissue stretched down to the pulsating dura mater seen through the large defect in the first sacral arch. This mass was dissected from the dura at the point where it found attachment. The dura was found to be not adherent to the overlying laminæ in the neighborhood of the hiatus. The dura was then incised, cerebrospinal fluid escaping. Within the dural opening several nerve roots were seen floating in the fluid, some lying against the inner surface of the dura, but it was demonstrated that they were not attached to it. The dura was sutured and the hiatus was closed by a plastic operation.

Microscopic examination of the removed mass of fat, connective tissue, and aponeurosis showed no other structures within it.

The patient vomited and had headaches for several days after the operation, and required catheterization. Primary union. Involuntary urination developed for a few days and transitory diarrhea recurred.

Sensory examination a few weeks after the operation showed some improvement in the left lower extremity, as indicated in Figs. 12 and 13. She was discharged August 22, 1908. During the next few months she complained much of pain in the back. The pains in the left leg had, however, gradually diminished.

In October, 1908, there developed a plantar ulcer surrounded by callus, which healed very slowly. During the next few months she complained much of severe pains in the left groin and the back. During the following year recurrence of ulcers in the sole of the foot and calf, always preceded and accompanied by fever.

Operation (Elsberg and Brickner), May 16, 1910. Lumbar laminectomy—first, second, and third lumbar laminæ removed. Dura opened and the exposed cord revealed the following: Its arteries were more prominent than normal. Much of the pia was lifted from the cord by coagulated cerebrospinal fluid. The cord proper extended farther down than usual, namely, to the third lumbar arch. The coagulated fluid was liberated, but no further lesion was noted in the cord or its roots, anterior or posterior, at this level. Laminectomy then continued down to the spina bifida, removing the fourth and fifth lumbar arches. There was then revealed at about the level of the fifth lumbar arch what appeared to be a lipoma of the cauda equina, occupying all of the spinal canal at this situation. It was soft, yellow, and of irregular conformation. Small pieces were removed for histological study, but it was impos-

sible to dissect the fat from the roots of the cauda equina, which ran through it and were intimately attached to it. The posterior fifth lumbar nerve root on the left side was then divided to relieve pain. Histological examination of this fatty tissue showed that it was a fibromyolipoma and contained a number of fine nerve bundles, some of which were apparently ramifications of larger nerves lying in the connective tissue. Most of the nerve bundles showed proliferation of the endo- and perineurium, and some of those lying in the fat were almost completely converted into fibrous cords. The connective tissue was rich in capillaries and arterioles, in most of which there were leukocytes, with perivascular aggregation of white cells. Some of the scattered striated muscle fibers showed degeneration. (Examination by Dr. I. Strauss.)

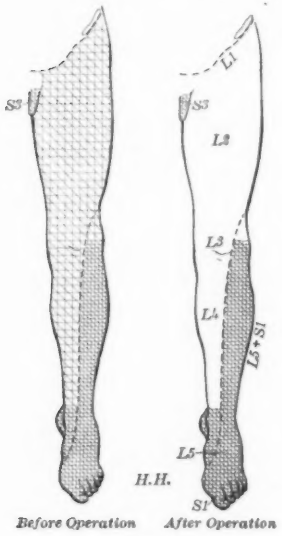

FIG. 12

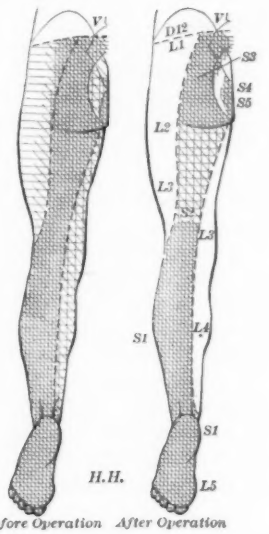

FIG. 13

Since this operation the patient's symptoms have progressed. A few months after it she developed drop-foot on the left side and subsequently also on the right side. She has very poor control of the bladder or rectum, complains much of backache, has cystitis from time to time, has frequent attacks of mucous colitis and frequent recurrences of the ulcerations. She walks but little, and out of the house is obliged to use a wheel chair.

These two cases are especially interesting in the laterality of the bifidity and, for a long period of years, of the symptoms. *They are interesting also in developing the observations concerning the*

growth of callus as a purely trophic phenomenon independent of pressure, and the prostration and marked rise of temperature which preceded and accompanied the outbreak of an ulceration, as we repeatedly noted.

Sam. S., male, aged thirty years, suspender-maker, was admitted to the dermatological service at Mount Sinai Hospital July 15, 1915, with an ulceration, about two inches by three inches in extent, on each buttock, symmetrically situated two and a half inches from

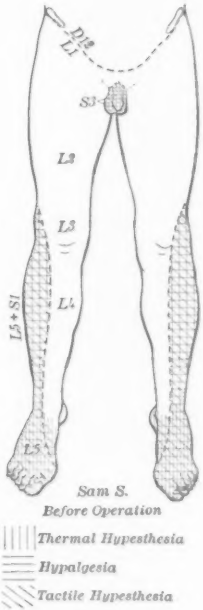

FIG. 14

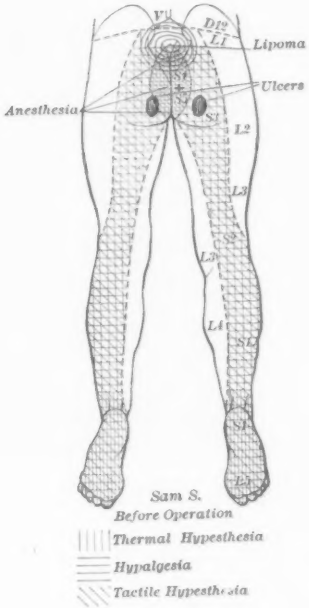

FIG. 15

the anus. These punched-out, freely discharging ulcerations had healed and recurred three or more times in a period of two years. The patient also had a purulent cystitis, of unknown origin, a right inguinal hernia, a peculiar falsetto voice, and a lipoma over the lumbosacral area characteristic of spina bifida occulta. He had no motor or rectal symptoms, but there was symmetrical sensory paralysis of the anoscrotal regions, buttocks, and lower extremities, as shown in Figs. 14 and 15. No alteration of the reflexes. Wassermann reaction negative.

A roentgenogram showed a narrow cleft in the fifth lumbar arch and an absence of all the sacral arches (Fig. 16).

Under local treatment in bed the ulcerations healed and the patient was discharged, under observation, August 27, 1915.

When he reported again, ten days later, the ulcerations had recurred as before. He was then referred to me for operation and admitted to Dr. Elsberg's service.

FIG. 16.—Sam S. Roentgenogram showing cleft in L5 arch and wide hiatus in all the sacral arches.

Operation (Brickner), September 10, 1915. Semicircular incision over the upper and left border of the mass, a baseball-sized flattened lipoma, characteristically congenital in its fine lobulation and absence of capsule. It was cautiously dissected out. The wide sacral dehiscence was found covered throughout with a dense aponeurosis except at its upper end, where a small sac protruded into the surrounding fat, to which it was adherent. This sac was found to contain a few nerve roots so adherent to its inner surface that they could not safely be dissected from it. The herniated mass was a little larger than a thimble and escaped through a finger-tip-sized opening in the spinal canal. Below this opening the aponeurosis was incised to permit exploration. Only fat was seen, extra- and intradurally. Some of this was removed. The protruding nerve roots and attached dura were reduced into the canal *en bloc* and the opening was closed over firmly by a plastic operation on the aponeurosis.

Nine days later the wound was healed *per primam*, the ulcers were almost healed, and there was a decided improvement in the sensory disturbances. This improvement has continued (Figs. 17 and 18), and today practically nothing remains of the sensory paralysis—formerly so marked in the anogenital and gluteal regions—except thermal hypesthesia of the feet. I operated upon this patient a year ago for his inguinal hernia, and I have kept him under observation since the spinal operation two years ago, during which time the buttocks have remained solidly healed. Six months ago he had an infected abrasion of the dorsum of the right foot, but it healed normally.

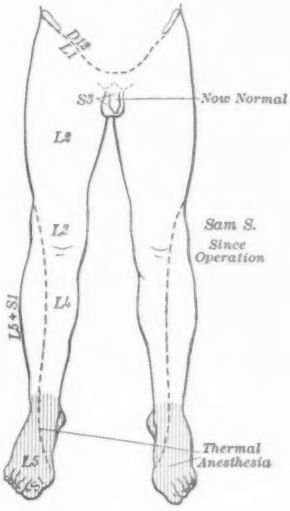

FIG. 17

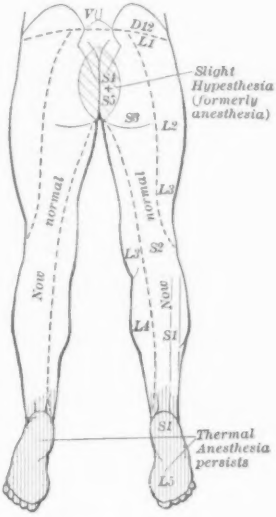

FIG. 18

Even if the continued cure of the previously recurring ulcers is a mere coincidence—which I cannot admit—the pronounced change in the sensory phenomena bespeaks a real benefit from his spinal operation.

GROUP II. *Spina Bifida Occulta, with External Signs, without Symptoms.* It would seem probable, especially from a study of the literature of abnormal hair growths, that some individuals with spina bifida occulta, even though marked by a very decided hypertrichosis or a congenital lipoma, go through life without any symptoms, or, at any rate, without any symptoms that bring them to the physician's attention. The following is such a case, although the patient died before he was twenty-three years of age.

Harold H., male, aged twenty-one years, was referred to me by Dr. Bullowa, and was in Dr. Elsberg's service at Mount Sinai Hospital in 1914 with a chondrosarcoma of the head of the tibia. Resection of the knee (Brickner), recurrence, amputation of thigh (Brickner). Intrathoracic metastasis. Died at Roosevelt Hospital, November 18, 1915. No motor, trophic, or sensory disturbances were noted, but he had a localized, bushy growth of long, straight hairs over the lumbosacral region (Fig. 19), and a roentgenogram showed a wide cleft in the fifth lumbar arch and a wide hiatus in the first, second, third, and fourth sacral arches (Fig. 20).

FIG. 19.—Harold H. Hypertrichosis over spina bifida occulta.

R. S., female infant, aged three months, admitted to Dr. Moschcowitz's service at Mount Sinai Hospital August 18, 1916, with a congenital lipoma over the lumbar spine, small at birth but gradually increasing in size to that of a large crab-apple. No defect in the vertebral arches could be felt through it, and pressure upon it caused no symptoms. Physical examination otherwise negative.

August 22. Operation for spina bifida occulta lumbosacralis (Brickner). The swelling was partly circumcised and the lipoma, finely lobulated and non-encapsulated, was cautiously dissected up. A cleft was palpated in two arches, probably L5 and S1, closed over

by a tough membrane (membrana reuniens) except at one spot, through which fat extending from within the vertebral canal became continuous with the extravertebral lipoma. The membrane was incised to permit exploration, exposing much white fat lying upon and among the posterior roots of the cauda equina, which appeared normal. Much of this fat was picked out and the herniated portion

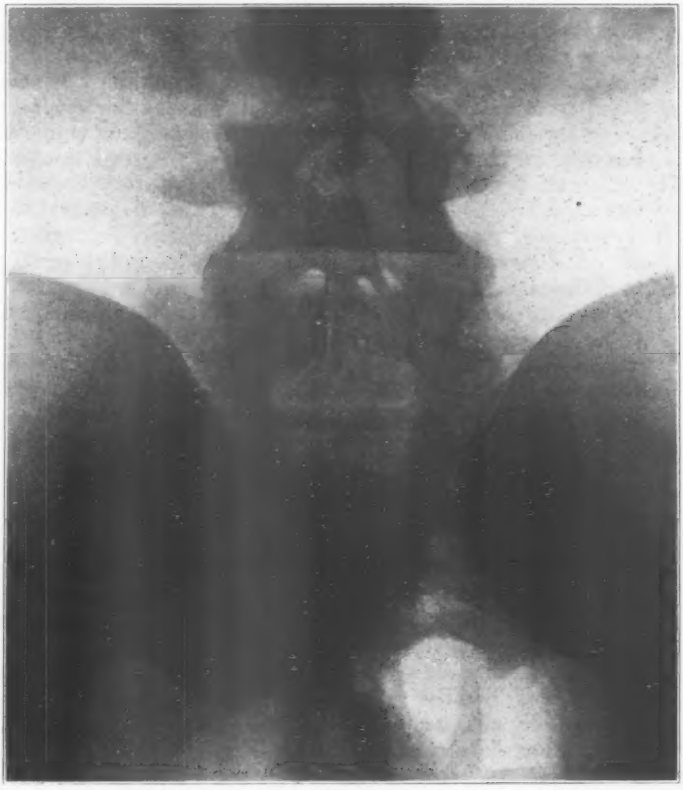

FIG. 20.—Harold H. Narrow cleft in L5 arch and wide hiatus in (almost complete absence of) S1 arch.

was removed. The membrane was closed with sutures, and a plastic closure of the lumbar aponeurosis was made over this. The baby was discharged, well, September 15.

Although the infant had had no symptoms the operation was done as a means of preventing, if possible, the development of symptoms later in life.

GROUP III. *Spina Bifida Occulta, without External Signs, with Symptoms.* This is the group to which I especially call attention, for it has hitherto scarcely been described.

In April, 1909, when I demonstrated before the surgical section of the New York Academy of Medicine the first and second cases here reported, the almost insignificant lipoma in the second case led me to conclude by urging "a careful scrutiny of the back and an *x*-ray picture in all cases in which there are incontinence of urine or feces or, in the lower extremities motor, sensory, or trophic disturbances, the cause of which is not otherwise evident."[6] I find that in the same year, 1909, Alfred Fuchs[7] published a study of 24 cases of enuresis in adolescents and adults, in which he described the following conditions, variously associated, as due to a congenital lower cord lesion which he called "myelodysplasia:" (1) Sphincter weakness, especially the so-called neurosis, enuresis nocturna. (2) Well-developed syndactyly between the second and third toes, and sometimes the fourth toe, in 66 per cent. of the cases. (3) Dissociated sensory disturbances in the feet, especially the toes, but also at the outer side of the feet, and chiefly thermal hypesthesia or anesthesia, in 75 per cent. (4) Defect in the sacral canal varying from an abnormally high opening of the hiatus sacralis to a spina bifida. Fuchs found these vertebral anomalies roentgenographically in 6 out of 10 cases of enuresis, and regards them as a rudimentary spina bifida occulta development. (5) Disturbances of skin and tendon reflexes, 11 of the 24 cases. (6) Foot deformities—percentage not stated.

Georg Peritz[8] agrees with Fuchs that the association of enuresis and spina bifida occulta sacralis is not merely accidental, thus disagreeing with Tromner and also with Lewandowsky, who, in his *Handbuch der Neurologie,* says that enuresis and spina bifida occulta are on the same degenerative basis, but have nothing to do with each other. In 22 adolescent cases of enuresis Peritz found spina bifida occulta roentgenographically in 68.2 per cent. In 20 children with enuresis Peritz found spina bifida occulta roentgenographically in 35 per cent.

William G. Spiller[9] reported a case of enuresis increasing, and motor weakness and sensory disturbances developing, after moderate exercise, in the lower extremities, in which the roentgenogram showed a grave defect of the sacrum and lower lumbar vertebræ, the back appearing entirely normal to sight and palpation.

Palmer Findley[10] renews attention to the fact that "virginal prolapse of the uterus occurs with greatest frequency in the newborn, and in most instances there are associated congenital deformities, notably spina bifida," and indicates that it would appear to be also a contributing factor sometimes in prolapse of the uterus in multiparous women

In 17 cases of congenital prolapse of the uterus Ebeler and

Duncker[11] record that 15 had spina bifida; and they found occult spina bifida roentgenographically in 23 out of 28 multiparous women with prolapsus uteri. They also found it in 3 of 28 multiparous women without prolapsus.

The following 3 cases may be cited to illustrate this group. Palpation may or may not give sufficient indication of spinal arch deformity or dehiscence. Roentgenography shows it clearly.

Mollie C., unmarried, female, aged twenty-one years, was referred to me by Dr. Leon Lesser in December, 1915, for a partial incontinence of bladder and rectum, which existed since she was nine years old. She talked when one year old but did not walk until four. When in the street she often soils herself with urine or feces; at home she is able to reach the toilet in time to prevent this mishap. Her chief complaint is that she therefore must remain indoors most of the time. She urinates five or six times daily and once or twice during the night. Usually she has no diarrhea.

There were no other symptoms. The urine was normal. Physical examination was entirely negative. I found no sensory, trophic or motor disturbances and no abnormality of the reflexes. Nevertheless it occurred to me as possible that, in spite of the absence of any external sign, there might be an occult spina bifida. (I was not then familiar with the articles of Fuchs and of Peritz.) A radiogram showed a cleft in the third and fourth sacral arches (Fig. 21).

I had the girl admitted to Dr. Elsberg's service at Mount Sinai Hospital, where he and I observed her for a week, but no new data were developed. Cystoscopic examination showed no bladder lesion. In this case the limited character of the symptoms did not appear to urge the desirability of a spinal exploration. There is obviously no hernia of the cauda equina. The lesion is perhaps in the cord itself. Her condition has not improved during the past year and a half, however, and perhaps an exposure of the cauda equina may reveal some roots adherent to the membrane across the cleft, the release of which may be beneficial.

Benjamin T., male, aged fifty-eight years, was admitted to Dr. Moschcowitz's service at Mount Sinai Hospital June 3, 1916.

Twenty-five or more years before he developed a plantar ulcer of the left foot—an infected wound arising, he thought, from a shoenail. The foot became much swollen and the ulceration remained unhealed for several years. The infection gradually involved the first phalanx of the great toe, which was removed. Two weeks later the fourth toe became involved and the first and second phalanges were removed. Over the finally healed plantar sore callus formed, which the patient frequently pared down. For thirteen years the foot remained healed. Then a perforating ulcer reappeared at the same plantar site, and was finally healed. It

recurred four years later and persisted again for a long time before healing was effected, after treatment at the Vanderbilt Clinic and Har Moriah Hospital. Six months later it again recurred and involved the third toe, which was removed by Dr. Moschcowitz. This was followed by healing.

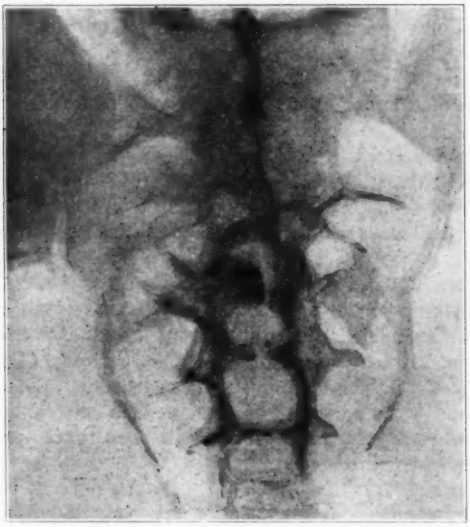

FIG. 21.—Mollie C. Sacral spina bifida occulta. Narrow clefts in the S3 and S4 arches.

Three months before admission to the hospital a painless blister, then an ulcer, appeared on the second toe, increased in size and depth, and extended into the metatarsophalangeal joint. The ulcer was the size of a dime, with sharp borders. The toe and the corresponding metatarsal region were dull red and swollen. There was slight tenderness. In a callous area over the ball of the foot was a linear scar 2 inches long. The fifth toe appeared normal; the first and the fourth had only the terminal phalanx; the third toe was missing; the second has been described.

The appearance was not that of arterial disease. The dorsalis pedis and posterior tibial pulsated normally on both sides. There was no "blushing" or "paling" phenomenon. The urine and heart were normal. Wassermann reaction negative. Blood-pressure 150 to 170. Emphysema. Gait and station normal. No bladder, rectal, or motor symptoms. The neurologist reported "a negative neurological examination. The condition does not depend upon any neurological disturbance."

It seemed to both Dr. Moschcowitz and me, however, that the phenomena were trophic in origin, and I suggested a roentgenographic examination of the lower spine for a bifidity. The sacral spinous processes were not palpable. Indeed, there appeared to be a depression involving the lower half of the sacrum. The roentgenogram showed an extensive sacral hiatus (Fig. 22). There is, in fact, thermal hypesthesia of both feet, although no apparent tactile hypesthesia or hypalgesia.

FIG. 22.—Benjamin T. Extensive sacral hiatus.

The third toe was amputated. With the healing of this wound there were no further symptoms and an exploration of the spine was considered unjustified. The patient is, however, under observation.

The following case I present quite tentatively, for the diagnosis is doubtful.

Max H., male, aged fifty-two years, on the neurological service of Dr. Sachs at Mount Sinai Hospital, January 17 to February

FIG. 23.—Max H. Symmetrical ulcerations of feet.

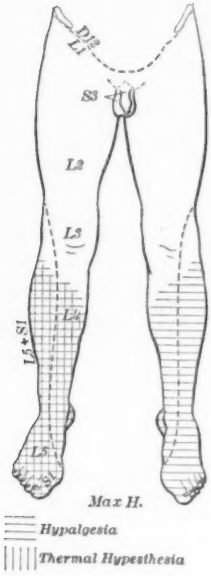

FIG. 24

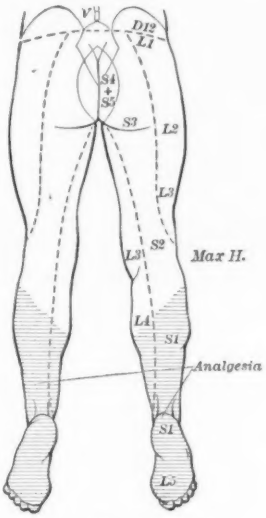

FIG. 25

24, 1917. Ten weeks before admission he had burning in the feet radiating into the legs, sometimes with tingling sensations in the finger-tips. Four weeks later ulcers developed on the feet, painless, and not tender. For some time he had been urinating very frequently, every half-hour during the day and twice at night; no shooting pains or trouble in walking.

FIG. 26.—Max H. Extensive sacral hiatus.

Examination gave the following positive findings: Hands cyanosed, cold and clammy; coarse tremor. The feet are cold, slightly cyanotic, perspiring. They show the following symmetrical ulcerations (Fig. 23): On each heel an area of dry gangrene about the size of a quarter; on the inner aspect of each big toe a gangrenous area the size of a dime; the right big toe shows also a gangrenous area the size of a nickel. There is ulceration about the nails of the second and third toes of the right foot. Pulsation of the dorsalis pedis artery is readily palpable in both feet. Hypalgesia and

analgesia are noted in both extremities, as shown in Figs. 24 and 25. Cerebrospinal fluid: 11 cells, 32 per cent. lymphocytes, 68 per cent. polynuclears; Wassermann reaction negative. Urine negative. Roentgenogram shows extensive sacral hiatus involving all of the arches except the first (Fig. 26). Electrical reactions normal.

The neurologists inclined to the diagnosis of a neoplasm either just above the conus or in the cauda equina. Dr. Sachs made the following note in the history: "Differential diagnosis between spinal gliosis with possible cavity and a neoplasm of the cauda equina. The onset thirteen weeks ago with sensory phenomena, rapid ulcer formation with gangrene, with preservation of gross muscular power and absence of marked vesical symptoms, argues in favor of neoplasm rather than gliosis. Even if gliosis were the condition, exploratory operation is advisable." Dr. Abrahamson believed that the lesion is a tumor of the type found in spina bifida occulta. Dr. Strauss seemed more inclined to think that the spinal bifidity is an accidental association and that the lesion is a cord tumor just above the conus.

February 17. The patient had a hemoptysis and developed dulness at the right apex. About the same time it was noticed also that there was diminished power in the right lower extremity. He refused operation and left the hospital unrelieved.

None of my 3 cases of spina bifida occulta without external signs with symptoms, and none of the cases of enuresis, etc., recorded by Fuchs, Peritz, Spiller (and one by Alfred Sänger[15]), has been submitted to operation, and a lesion of the cord structures corresponding in type to a spina bifida has therefore not been demonstrated.

Admittedly in some cases at least the bifidity of the spine may be a congenital abnormality that has no etiological relationship with the symptoms, but in many cases, and especially in the case of Benjamin T., with the fairly characteristic progressive ulceration of the toes of one foot and the thermal hypesthesia of both feet, the presumption is very strong that the lesion at fault is indeed a spina bifida occulta.

Group IV. *Spina Bifida Occulta, without External Signs, without Symptoms.* For some years I have been interested in observing that a considerable number of roentgenograms of the lower spine of patients exposed for examination of the urinary tract, the hip, etc., shows a cleft most often in the fifth lumbar arch or in one or more of the sacral arches. In what percentage of apparently normal individuals this anomaly is found roentgenographically I cannot say, but I have the distinct impression that it is not very uncommon. Perhaps a careful inquiry into some of these cases might show some disturbance of bladder control or loss of thermal or tactile sensitiveness in the lower extremities; but in the absence

of such a study I think we may be prepared to believe that there is found in a certain proportion of healthy individuals a congenital defect in the lumbar or sacral arches without any affection of the spinal cord tissues. The following cases are illustrative.

FIG. 27.—George K. Asymmetry of L5 arch, cleft in S1 arch, wide defect in lower sacral arches.

The first is that of a spina bifida occulta involving the first and the lower sacral arches (Fig. 27), probably not associated with the symptoms:

George K., aged forty-six years, was in the neurological service of Mount Sinai Hospital January 17 to January 31, 1917, with acroparesthesia of the hands and feet.

Following a fall on his back he had suffered for ten years with radiating pains in the lower extremities, varying with the weather. For ten months he had continuous burning pain in the tongue, hands and feet. His hands and feet swelled when dependent.

He had paresthesia ("pins and needles") in the feet. For five years he had impotence and prostatorrhea.

There was decided hyperesthesia of the legs, anteriorly and the buttocks. Tendon reflexes of all four extremities much exaggerated. Lumbar puncture—normal cerebrospinal fluid. Slight polycythemia. Palpation of the sacrum suggests an absence of the lower spinous processes. Other data negative. Roentgenogram shows a spina bifida occulta, illustrated in Fig. 27.

The following case is interesting in the differential diagnosis of the ulceration and gangrene of the toes, the peculiar anomaly of the lower vertebral arches, and in the operative finding:

Isidore B., Russian, male, aged twenty-eight years, was admitted to Dr. Beer's service at Mount Sinai Hospital in August, 1916, with moist gangrene of the fourth and fifth toes of the right foot and mild cellulitis of the anterior portion of the foot. For two years there had been progressive infection, ulceration, and gangrene of the third, second, and first toes of this foot, with failure of the stumps to heal and extension of the process to the plantar surface. The involvement of the fourth and fifth toes had begun three months before admission. During all this time he suffered *severe pain*, which is characteristic of thrombo-angiitis and not of the trophic gangrene of spina bifida.

On the left side the dorsalis pedis and posterior tibial arteries pulsated well, but on the affected side no pulse could be felt in either vessel. The superficial and deep reflexes of all extremities were exaggerated, especially in the lower extremities. No sensory disturbances were elicited. Wassermann reaction negative.

Dr. Abrahamson noted a superficial cleft in the tips of the spinous processes of the eleventh and twelfth dorsal and first and second lumbar vertebræ. A roentgenogram revealed the peculiar deformity of the fifth lumbar and first sacral arches shown in Fig. 28. Although there is no actual cleft in any arch, this type of lamina malformation is seen, as I have shown, in other cases with bifidity; it produces a distinct hiatus vertically, and I think the abnormality may be properly grouped with spina bifida occulta.

Although the patient's disease was fairly diagnosticable as a thrombo-angiitis obliterans, it seemed worth while to give him the benefit of a doubt suggested by the vertebral deformity and to explore it for a possible cauda equina lesion. Dr. Buerger kindly transferred the patient to my care for that purpose.

Operation (Brickner) September 8, 1916. The fifth lumbar and first sacral arches were exposed and found abnormally separated. They were removed, the dura was incised, and the cauda equina was exposed in its bed of fat. It presented no gross lesion. The dura appeared in all respects normal. Spinal fluid escaped under great pressure.

The wound healed *per primam*, and after the operation there was a remission in the subjective symptoms. The second toe was

amputated September 19. There has been a progression of the disease, and the foot will probably require amputation.

The amputated toe showed "chronic suppurative inflammation, granulation tissue, and endarteritis. A diagnosis of primary arterial lesion cannot be determined from the specimen" (Buerger).

FIG. 28.—Isidore B. Malformation of L5 and S1 arches producing a longitudinal hiatus.

Taken all together, the results of operation for spina bifida occulta are not brilliant, and probably for the reason that the degenerative and neoplastic processes are scarcely remediable. Those cases in which there is a hernia of the spinal roots probably offer the best chance for a good result; for the reduction of this hernia into the bony canal, by relieving the contained nerve roots of traction and pressure, may greatly improve the existing symptoms or prevent the development of some others. The third case, Sam S., the young man with the trophic ulcers of the buttocks, represents, I think, a highly satisfactory result from such an operation. Even when there is no meningocele much may be found possible at operation—for example, separation of adherent nerve roots from the membrana reuniens, division of a constricting band, removal

of an endostosis, or *perhaps* even the removal of a teratoma of the cauda equina.

I believe that the following are legitimate indications for operation:

1. In infants and children, spina bifida occulta with congenital lipoma or hypertrichosis, even though without any symptoms—to reduce the spinal hernia into the canal or to meet any other indication that is found, in the hope of *obviating* the development of symptoms during adolescence. The case of the infant that I have reported is illustrative of this indication.

2. In adults, spina bifida occulta with sufficiently serious and especially with progressive symptoms, and this I believe should apply whether or not the spina bifida occulta is marked by external signs (lipoma, hypertrichosis). I am not unmindful of the inherent risks of such an operation, but surely progressive gangrene of the lower extremities and incontinence of the sphincters are indications sufficient to justify the taking of such risks. In the literature I have found records of 12 operations. I am adding now 5 to these—*all of the 17 without mortality*, and some with decided benefit. In all 5 operations for spina bifida occulta within my personal experience there was no untoward result, and the wounds in all cases healed *per primam* and without any spinal infection.

In making this presentation of a condition that has for so many years interested me, it has been my purpose to call attention to the four groups into which it seems to me we may clinically divide spina bifida occulta, and to suggest the advisability of a roentgenographic examination for such a condition in all cases of sphincteric, sensory, trophic, or motor disturbances that might thus be explained, even though there is no external manifestation to suggest its presence.

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DIATAXIA CEREBRALIS INFANTILIS: THE ATAXIC TYPE OF CEREBRAL BIRTH PALSY.*

By J. Ramsay Hunt, M.D.,

CONSULTING NEUROLOGIST TO THE NEUROLOGICAL INSTITUTE; THE NEW YORK HOSPITAL, BABIES' HOSPITAL, RANDALL'S ISLAND, AND THE LETCHWORTH VILLAGE FOR MENTAL DEFECTIVES; ATTENDING NEUROLOGIST TO THE MONTEFIORE HOME AND HOSPITAL, NEW YORK.

The classification of the cerebral palsies of early life has engaged the attention of neurologists and psychiatrists for a number of years. A great many clinical types have been described, and in recent years there has been considerable progress in our knowledge of their various causes and underlying pathological changes. The subject, however, still presents many obscurities in etiology, localization and symptomatology.

The cases have been variously grouped in accordance with the etiology, the nature of the pathological changes or the clinical symptoms presented. For all practical purposes the clinical classification is the important one and the most useful, as post-mortem examination and histological study are usually necessary to determine the true pathology of any case and the original causative factors are often obscure and difficult to determine. This is especially true of the congenital cerebral defects and malformations resulting from disease of the fetus during the period of intra-uterine life.

The common clinical type of infantile cerebral palsy is the spastic form. This may appear as hemiplegia, paraplegia or triplegia, and very frequently as a cerebral diplegia. It is characterized by paralysis with various spastic phenomena, either alone or in association with other cerebral symptoms, such as choreiform manifestations, ataxia, tremor, athetosis, mental defect, epilepsy and disturbance of the special senses.

A classical description of this large clinical group and its relationship to injuries received at birth was given by an English physician, Dr. Little, in 1862. In recognition of this important contribution, those cases of *cerebral spastic paralysis* resulting from injury to the brain at birth are very commonly known under the generic title of Little's disease. Little's conception that certain abnormal types of parturition, especially those associated with difficult and prolonged labor, produced hemorrhages over the surface of the brain which interfered with the normal functions and development of motility has been upheld by many subsequent investigators. Prominent among these may be mentioned the names of Sarah McNutt, Freud, Gowers, Railton and Kundrat.

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Little also pointed out the importance of premature birth as a cause of cerebral spastic paralysis, and some observers, notably Brissaud, would limit the term Little's disease to this comparatively small group of cases, in which the development of the pyramidal tracts has been checked or retarded by premature delivery. This limitation of Little's conception in the opinion of many writers is not justified, and a perusal of his original monograph will, I believe, convince most readers that the essential idea underlying his work was the relationship of all forms of abnormal birth to the cerebral spastic palsies of early life.

Förster, in 1909, described in some detail an unusual clinical type of cerebral diplegia which he termed the *atonic-astasic type* of infantile cerebral palsy. This group stands in striking contrast to the spastic type of Little, and is characterized by motor paralysis with flaccidity. There is inability to stand or walk (astasia-abasia), great difficulty in articulation, not infrequently mutism, and a marked mental defect. A certain degree of individual movement of the extremities is possible, but all the larger and more complicated motor activities are impossible, owing to a defective action of antagonistic and synergic groups of muscles. Slight spastic phenomena are sometimes noted in the lower extremities, but the essential quality of the paralysis is flaccid and atonic.

Many of the symptoms in this group of cases suggest a cerebellar origin, and Förster was at first inclined to attribute this origin to the symptomatology until later two of his cases subjected to postmortem examination revealed an extensive lobar sclerosis of the frontal region, the cerebellum appearing quite normal on macroscopic examination. Unfortunately, no microscopic studies were recorded.

A few years later, in 1913, Pierce Clark reported similar cases under the rather comprehensive title, "Cerebrocerebellar Diplegia of Flaccid Atonic Type," and concluded on purely clinical grounds that the lesion was probably a combined one, involving both the cerebrum and the cerebellum, notwithstanding the negative cerebellar findings in Förster's cases.

Clark assumed that a cerebellar lesion alone would be amply compensated for by the cerebrum if the cerebral structures were normal, and therefore a combination of cerebral and cerebellar symptoms as occurs in this group of cases would be in favor of a combined lesion of these two structures. Against this point of view, however, may be mentioned the normal appearance of the cerebellum in Förster's cases and also the fact that grave disturbances of muscle tone and coördination may result from lesions of the cerebral cortex. Accordingly, the term cerebrocerebellar diplegia in the present state of our knowledge would seem hardly justified and the simple clinical designation atonic or flaccid type of cerebral diplegia as used by Förster is, I believe, preferable.

Batten and von Wyss have also recorded examples of this flaccid type, and there can be no question as to its distinctive clinical characteristics and marked contrast to the spastic types of Little.

Under the title "Congenital Cerebellar Ataxia," Dr. Batten has described what he terms a pure *cerebellar type* of diplegia. This group is characterized by ataxia of the cerebellar type, which displays, however, a decided tendency toward recovery. There is little or no mental defect, and the clinical picture is one of pure ataxia of cerebellar origin involving speech, the gait and station and various movements of the extremities.

Therefore, according to our present classification, the cerebral diplegias of childhood may assume one or other of the following clinical forms; the *spastic* type of Little, the *atonic* or *flaccid* type of Förster and a *cerebellar* type as described by Batten. These may exist as such in pure form or may occur in various combinations. They may also present a variety of complications and associated symptoms such as epilepsy, mental defect, choreiform and athetoid manifestations, which give to this whole chapter a large and varied symptomatology.

The group of the cerebral diplegias to which I would direct attention as a distinct clinical variety is an *ataxic type of cerebral birth palsy*. The ataxia is bilateral in distribution and is characterized by a generalized disturbance of coördination involving the gait and station, the use of the arms and legs, and the muscles subserving the function of speech. The incoördination persists in the recumbent posture and has the characteristics of a true ataxia. It is, I believe, of cortical origin. There is neither paralysis nor spasticity, and the cases observed were free from serious mental defect or epilepsy. In the absence of any direct pathological evidence I would suggest, as the probable etiological factor, an injury to the fetal head received at birth, producing rupture or thrombosis of the cerebral veins, with limitation of the lesions to the cortex of the parietal lobes.

The clinical picture is one of pure *cerebral diataxia*, and may be regarded as the sensory equivalent of *cerebral diplegia*. In my opinion, however, the vascular lesion, instead of being limited to the motor area as in spastic diplegia, is situated more posteriorly behind the fissure of Rolando in the sensory sphere of the cortex, thus implicating the centers and commissural systems which are engaged in the reception and transmission of the memories of movement.

The parietal lobe is the great receptive center for the registration of sensory stimuli, representing movements and the various combinations of movement, and a bilateral lesion in this area would necessarily impair in greater or lesser degree the normal development of motility and coördination.

CASE I.—The patient, E. C., was a boy, aged twelve years, born of healthy parents. There was no history of any familial nervous or mental disease. With the exception of measles, chicken-pox and tonsillitis there was no history of any serious illness.

He was an only child, born at term, the labor being unusually difficult and prolonged. The mother stated that she was thirty-five years old at the time and was in labor for forty-eight hours. The membranes had ruptured early and delivery was eventually effected by forceps. The child was born blue and asphyxiated and did not cry for several days. There were general convulsive seizures during the first few days, but none subsequently. The head was said to have been out of shape and deformed, but gradually assumed a normal symmetry. An effort was made to feed the child by the breast, which was unsuccessful and had to be abandoned.

Very early in the child's life it was noted that the movements of the extremities and trunk were weak and awkward. Swallowing was performed without apparent difficulty; there was, however, a tendency to drooling, which persisted for several years. It was noted that the child did not sit up or show much tendency to creep until the end of the second year. The movements then were awkward, unsteady and accompanied by unusual efforts. Unsteadiness of the movements was noted quite early and has persisted up to the present time. Both sides were involved, but the right arm seemed to be more affected than the left, and the legs showed a greater degree of disability than the arms.

The child did not speak until the third year, and then only uttered inarticulate sounds. He has shown slight but steady improvement from year to year in speech, in his ability to sit or stand and in the use of the arms. He could not stand alone until the sixth year, and is still unable to walk alone.

The general health and intelligence are both good. The mother insists that the child is bright and learns readily. He is quite cleanly in his habits, and is able to control the sphincters. He is not able to feed himself or dress himself on account of the severe motor ataxia. He is good tempered, and is interested in the books and games suitable to his age. He has had no convulsions since the initial spasms after the birth, which lasted several days. The special senses are apparently not affected.

Examination, May, 1916. The boy is of good size, well nourished, and appears in excellent general health. The muscles are moderately well developed. There is a slight tendency to motor restlessness and atactiform movements unless supported and in a relaxed position. This unrest is increased by any action, however slight, and by mental excitement. There is no true chorea or athetosis, but the ataxic disturbance has a somewhat choreiform character when the motor agitation is extreme.

He is able to stand by holding on to a chair, but not unsupported. While standing in this position there are swaying, unsteady movements of the trunk and arms in the effort to maintain the equilibrium. The head also shows ataxic oscillations. On attempting to take a few steps, even with assistance, the ataxic disorder becomes extreme and he falls backward or to the side and has to be supported; occasionally the legs give way under him.

In testing the movements of the upper extremities a very marked incoördination is demonstrable. On attempting to carry the finger to the tip of the nose there are irregular atactiform movements of the extremity and compensatory or associated movements in other parts of the body—for example, the head and trunk. This disorder of motility does not resemble an intention tremor but is a gross disturbance of the power of coördination. It is especially well marked if an effort is made to grasp a small object or to draw a line with a pencil. Such movements are carried out in a brusque and awkward manner, and any breakable object would surely be sacrificed if he were allowed to reach for it. If the attempt is made to hold out the arms with the hands and fingers extended there are irregular atactiform movements and changes in the relative position of the extremities, which are somewhat increased by closure of the eyes.

The alternating rhythmical movements of the wrists or fingers, as a result of the ataxic disturbance, are impaired. Dysdiadokokinesis is therefore present.

In the recumbent posture the lower extremities show a similar atactiform disturbance. If the attempt is made to place the heel upon the opposite knee there is a marked incoördination and uncertainty of movement, as might be observed in an advanced case of spinal ataxia.

Similarly, if the legs and arms are elevated while lying flat on the back and the effort is made to fix them in this position the extremities are moved about in extreme incoördination, and frequently the balance is lost and he rolls over to one side. The right arm and leg showed a somewhat greater degree of involvement than the left, which coincided with the early history and observations of the mother.

There is no demonstrable weakness or paralysis of the limbs. Indeed, the gross motor power of the lower and upper extremities is well preserved, except as modified by the ataxia. There is no atrophy. There is not the slightest evidence of spasticity in either the upper or lower extremities. Instead, the tonus of the musculature is moderately diminished.

The knee-jerks are present, equal and not exaggerated. The Achilles-jerks are inactive and only elicited with difficulty. The supinator jerks are not elicitable. The biceps- and triceps-jerks are inactive and variable. There are no periosteal reflexes in either the

upper or lower extremities and no clonus or spastic phenomena. The abdominal, cremasteric and plantar reflexes are present and of normal intensity. The Babinski phenomenon is absent.

The sensation, both superficial and deep, is apparently undisturbed. Touch, pain and temperature are recognized promptly, and so far as could be determined were well localized. The deep sensibility also appears normal. Pressure of the muscle induces pain and the sense of position is preserved. He recognizes the direction of passive movements of the toes, fingers and of the larger joints, promptly and without error. The sense of vibration is acute.

The pupils are equal and react promptly to light and accommodation. The pupillary skin reflex is active.

There is no nystagmus and the optic nerves are normal. On attempting to fix an object with the eyes there are slight rolling movements of the eyeballs, apparently of an ataxic nature, and associated atactiform oscillations of the head. There is no paralysis of the cranial nerves. The tongue is protruded in the median line, the palate is promptly innervated and the movements of the jaw are normal in power and extent. Facial innervation is equal on the two sides.

There is a considerable degree of dysarthria, and it is rather difficult to understand the spoken words. The mother, however, interprets perfectly and can carry on a very intelligent conversation with the boy. He can name ordinary objects placed in the hands, and there is no astereognosis. Sight, hearing and smell are normal.

The heart and lungs are normal.

The urine is free from albumin and sugar. The Wassermann test of the blood is negative.

NOTE.—The above findings were confirmed at several subsequent examinations. The essential disturbance was one of incoördination without paralysis or evidences of spasticity, which affected nearly all voluntary movements. While in the recumbent posture, with the musculature relaxed, there were no evidences of chorea or athetosis. On making the effort to stand or walk, and in certain postures, there were incoördinate movements which suggested a choreiform tendency, which, however, appeared to be dependent upon the degree of the ataxic disturbance. The right side showed a greater degree of involvement than the left. The tonus of the muscles was moderately reduced. There was, however, no undue flexibility of the extremities as occurs in the higher grades of hypotonicity. The knee-jerks were readily elicitable and not exaggerated. The Achilles-jerks could usually be elicited, but with difficulty, the same being true of the reflexes of the upper extremities. The plantar reflex was always of the normal type, and a true Babinski reflex was not obtained.

So far as could be determined the general sensations, both superficial and deep, were well preserved.

CASE II.—The patient, L. H., was a girl, aged six and a half years. The family history was good and there was no tendency to hereditary or familial diseases. The parents were healthy and were not related. The mother had given birth to two other children without special difficulty, and there had been no miscarriages.

The patient was the third child and the pregnancy had progressed without untoward complications. The birth occurred at term and the labor was extremely slow and tedious. After twelve to fourteen hours a faulty presentation was discovered and the delivery was effected by means of forceps. The child was born asphyxiated, and it was feared at the time would not survive. Artificial respiration was necessary and the child did not cry or take the breast for about two weeks. After this she took the breast, but only with some difficulty, and the cry was weak and feeble. The subsequent development was slow and retarded. The child could not sit up until two years of age, and then was unsteady and swaying, often falling backward or to one side. About this time she was able to creep, but with difficulty. Movements of the arms and legs were awkward and unsteady. Attempts to stand were not successful until the third year, and then only by holding on with the hands and accompanied by swaying of the body and frequent falls. Attempts at walking were only possible in the latter part of the third year, and then required careful assistance, as falls were frequent and often severe.

At the age of three years the child could speak a few words, but indistinctly, and only the mother could understand them. Swallowing occurred without difficulty and the special senses were apparently normal. The child was bright and took an interest in her surroundings, but was backward as compared with other children at the same age. On attempting to grasp an object with the hand the movements were uncertain and awkward.

The child had shown decided but gradual improvement. She had acquired better control of her movements and articulation was more distinct. She was fairly intelligent for her age but still somewhat backward in many things, which the mother attributed to her physical disabilities. There was no history of convulsive seizures.

Physical Examination October, 1914. The child is well nourished and fairly well developed for her age. While sitting quietly in a chair the extremities and trunk are motionless. There is a slight tendency to swaying and oscillation of the head, especially with any movement, such as fixation of an object with the eyes or when speaking.

Any attempt to raise an arm or leg evokes awkward ataxic movements of an incoördinate character. On attempting to grasp an object with the hands there is a series of irregular, incoördinate movements; the fingers are overextended, the hand is thrown about in an awkward manner and the object is grasped brusquely. There is no weakness of the movement or of the grasp—it is a pure ataxia of movement or incoördination. The disturbance is about equal on the two sides.

The child understands spoken language and talks, but articulation is dysarthric and indistinct and accompanied by slight atactiform movements of the head and trunk, especially if standing. In standing there is manifested a distinct disturbance of equilibrium. She stands on a broad base, with swaying of the trunk and various compensatory movements of the arms. This static ataxia is increased slightly by closure of the eyes. On attempting to walk a definite disturbance of the gait immediately becomes apparent. There is unsteadiness of equilibrium, pitching to and fro and great uncertainty of movement. In appearance it suggests very strongly the titubating gait of cerebellar disease. The legs are held far apart, the head erect and the arms used to balance. The movements of the legs are awkward and ataxic. On attempting to stand quietly there is a slight choreic unrest, which, however, is ataxic in origin and is not noticeable in a quiet and relaxed position. The ataxia does not disappear in the recumbent position. For example, on attempting the finger-nose test or the heel-knee test, while flat on the back, the incoördination of movements is scarcely less marked than in the erect posture or sitting in a chair. There is no cessation or diminution of the ataxia when the recumbent posture is assumed. In this position, if the arms and legs are elevated, and the attempt made to hold all four extremities upright, with the eyes closed, incoördinate, ataxic movements persist with unabated vigor. If the hands and arms are held outstretched, while sitting in a chair there are ataxic movements of the upper extremities and some swaying movements of the trunk. Rhythmic alternating movements of the wrist and fingers are performed slowly and awkwardly. Evidences of dysdiadokokinesis are therefore present.

The cranial nerves are normal. The tongue is protruded in the median line, the facial innervation, movements of the soft palate and of the mandible are all normal. These movements are associated with slight atactiform manifestations of the head and trunk.

The optic nerves are normal. There is no nystagmus and the pupils are equal and react to light.

There is no paralysis and all movements show a normal degree of power. The muscles are well developed and show no atrophy.

There is no spasticity in either the arms or legs and no clonus. The tonus of the muscles is slightly reduced but the hypotonicity is of moderate degree. There is plantar flexion of the toes; the Babinski reflex is not present.

The knee-jerks are present, equal and of normal intensity. The Achilles-jerks are faint and at times not elicitable with certainty. The arm reflexes, supinator, biceps- and triceps-jerks, are inactive and not definitely elicitable. The abdominal reflexes are present.

General sensation was apparently normal. Touch, pain and temperature stimuli were correctly interpreted and, so far as could be determined, were well localized.

The deep sensibility and sense of posture were also preserved.

The child could recognize the movements of the toes whether up or down, but if the order was given to place the left limb in the same position as the right, either the arm or leg, the attempt was made and sustained awkwardly and in an uncertain manner, probably on account of the ataxia. Sight, hearing and smell were not affected. There was no astereognosis. Such common objects as a coin, penknife or a key were promptly recognized in either hand.

There was no special mental defect; the child was good natured, responsive and well behaved and was not far below the mental average for her age.

Heart and lungs were normal. Wassermann test of the blood was negative. Urine negative.

NOTE.—This case, like the preceding, presented symptoms of a pure ataxic disturbance which affected nearly all voluntary movements, such as standing, walking and the individual activities of the extremities. The gait was very suggestive of a cerebellar ataxia but both the upper and lower extremities showed a definite ataxia which persisted in the recumbent posture. Lying flat on the back, with no demands upon the function of equilibrium, it was impossible to hold the extremities quietly in an elevated position as in cases of pure cerebellar disease, the ataxic disturbance persisting as in a case of spinal ataxia. There was no demonstrable weakness of the extremities and no spasticity. The sensibility was normal. There was no true chorea, athetosis or intention tremor. The slight choreiform tendency did not suggest true chorea but resembled rather the choreic unrest which is sometimes present in cases of advanced Friedreich's ataxia. The disorder of motility was an inability to regulate and coördinate voluntary movement. The condition was manifested soon after birth and was characterized by steady improvement. There was therefore no tendency to progression or aggravation of symptoms.

CASE III.—The patient, A. H., a girl, aged eleven years, was the last of seven children. The circumstances attending the birth were abnormal, the membranes ruptured quite early and labor was unusually prolonged and severe. The child was eventually delivered with forceps in an asphyxiated condition and was resuscitated with difficulty. She was said to have been "blue" for several hours after birth and the imprint of the forceps remained over the temporal region for a week or more. She did not take the breast readily and was nourished by the bottle. During the early months of life she was restless and difficult to quiet, crying a great deal, and was physically frail.

It was soon observed that the motor development was not normal. Such acts as holding up the head, sitting and attempts to crawl and walk were all considerably delayed, as was also speech.

She was unable to stand before the sixth year and then only with

assistance. The movements of the arms were noted to be irregular and unsteady early in the second year and articulation was indistinct up to the fourth year, but has shown a steady improvement since that time.

With the exception of summer diarrhea and chicken-pox the child has had no illnesses of note. The other children of the family were in good health. The father died of pneumonia and no history of any neuropathic heredity could be elicited.

The mother states that while somewhat backward for her age the child is bright, has learned to read a little and is especially fond of music. The special senses were not affected. There has been a gradual improvement in the motor disability from year to year, especially in speaking and walking. There has been no history of any convulsive seizures, excepting immediately after the birth, when there was an indefinite history of several spasmodic attacks.

Examination, November, 1912. The patient is thin and somewhat pallid; the teeth are uneven and defective.

The skeletal development is rather slight and fragile. The muscles are well formed and, although small, are not atrophic. On attempting to rise from a sitting posture there is a very evident disturbance of gait and station. She stands on a broad base, the trunk swaying from side to side and balancing with the arms. After a few moments she becomes somewhat adjusted to the change of posture, but slight atactiform and balancing movements persist. Closure of the eyes increases somewhat the degree of static ataxia.

While walking there is apparent a marked disorder of movement. The legs are lifted and thrown out in an awkward, ataxic manner and there are numerous compensatory lurches and balancing movements of the head, trunk and arms. She is, however, able to walk alone and with a fair degree of security, and does not fall. While sitting in a relaxed posture in a chair the musculature is quiet, and only occasionally slight atactiform movements of the head or arms are observed. On attempting to stand or walk or on initiating any movement of the extremities a considerable degree of ataxia results which is somewhat choreiform in appearance, but occurs only as an accompaniment of movement.

There is a general condition of marked incoördination. On attempting to grasp an object with the hand or on placing a finger on the tip of the nose a considerable degree of ataxia of the arms is apparent, which is increased by the associated atactiform movements in other regions of the body, as, for example, the head and trunk. The two arms are about equally affected.

There is also marked retardation and awkwardness in performing alternating rhythmic movements of the wrist and fingers (dysdiadokokinesis). There is no weakness of the upper extremities and the gross motor power is apparently normal.

The supinator reflexes are present and not exaggerated. The

biceps- and triceps-jerks are not elicitable. The muscles are somewhat relaxed and free from any spastic phenomena.

On attempting to hold the outstretched arms and hands at rest there occur slight ataxic movements which are increased by closure of the eyes. Objects placed in either hand with the eyes closed are promptly recognized and the stereognostic sense is unimpaired. The direction of passive movements of the fingers, wrist and elbow are promptly recognized with the eyes closed. With the eyes closed an attempt to place the segments of the upper extremity on one side in the same position as the other cannot be accomplished, owing to the ataxia.

The gross motor power of the lower extremities is well preserved and there is no special atrophy of the musculature. Passive movements are quite free, the muscles are relaxed and there are no evidences of spasticity. There is, however, marked incoördination of movement in the recumbent posture. The attempt to place the heel upon the knee or to elevate the extremity in a straight line produces a marked irregularity and incoördination of movement, which is sometimes accompanied by slight atactiform jerking movements of the head, trunk or upper extremities. The knee-jerks are elicitable and about equal on the two sides. They are not exaggerated and the responses are rather subnormal. The Achilles-jerks are faint and not always elicitable. The plantar reflexes are of the normal type (no Babinski).

Lying perfectly quiet and in a relaxed posture no choreiform or athetoid movements are present. If the arms and legs are then simultaneously elevated and the attempt is made to hold them in this position a great disorder of coördination immediately results usually with loss of balance and a tendency to roll over. The abdominal and epigastric reflexes are present and active on both sides.

The motor cranial nerves are normal. There is no paresis of the ocular, facial or palatal musculature. On protrusion of the tongue there are slight atactiform movements of the head and trunk, and in a lesser degree on testing the facial innervation or on fixing an object with the eyes.

There is no nystagmus. The pupils are equal and react to light and accommodation. The corneal and conjunctival reflexes are active. The jaw-jerk is not elicitable. Articulation is thick and indistinct. This is an ataxic, dysarthric disturbance of speech, and to the untrained ear is very difficult to understand.

There is no forced laughter, but the act of laughing is accompanied by ataxic movements of the head and trunk and to some degree of the extremities. The acts of deglutition and mastication are also accompanied by slight ataxic disturbance.

The special senses are not affected. Vision, hearing, and the sense of smell are normal. The optic nerves are normal.

The general sensibility is apparently undisturbed. Tactile, pain and temperature sensations are normal, and their localization, so

far as could be determined, was fairly accurate. The posture sense of the upper and lower extremities was apparently preserved and upward and downward movements of the joints were promptly recognized.

Heart and lungs are normal. Urine negative.

Mentally the child was bright and responsive and carried out the various tests promptly and intelligently. She was able to spell and could read short sentences, and the mother stated that she was making steady progress. She was cleanly in all of her habits and has had good control of the sphincters since early childhood. There has been a marked improvement in the child's condition during the past few years, but she still required constant care and supervision because of the general motor disability.

Resumé of the Clinical Reports. The cases which have just been described are very similar in their etiology and general clinical characteristics and constitute, I believe, a well-defined clinical type of cerebral birth palsy. They represent an ataxic type of cerebral diplegia which stands in striking contrast to the spastic type of Little and the flaccid or atonic type of Förster.

In all of the cases there was a history of severe injury at birth followed by retarded and defective development of the power of motor coördination. There was no paralysis, and if such existed after birth it was of a transient nature and the gross motor power was eventually completely restored. There were no evidences of spasticity. The active and passive movements as well as the reflexes revealed none of the characteristic signs of pyramidal tract involvement. The tonus of the muscles was slightly reduced and the tendon reflexes showed a corresponding reaction, and were moderately diminished and difficult to elicit. There was no undue flexibility of the extremities as occurs in the severer forms of hypotonia.

The essential symptom was a generalized ataxic disturbance affecting the gait, station, articulation and the use of the individual members. This was present in all positions, including the recumbent posture, and I would particularly emphasize the persistence of an ataxic disturbance of the arms and legs in the recumbent posture when no effort was required to maintain the equilibrium. The ataxic disturbance was also increased by closure of the eyes, by mental excitement and by any effort to carry out precise and definite movements.

The disturbance of coördination was bilateral and fairly symmetrical, but one side may show a greater degree of involvement. It affected the finer movements of the hands and fingers as well as movements of the larger joints, and seemed to be equally distributed in this respect. There was, at times, a slight tendency to motor unrest, somewhat suggestive of a choreic disturbance, which I regard as a manifestation of ataxia and the effort to maintain posture. The disorder of motility was clearly ataxic, and was

characterized by an inability to carry out precise and definite coördinate movements. It was not in any sense a true chorea or athetosis, the restless atactiform movements in certain postures merely suggesting the choreiform tendency. Dysarthria was also a well-marked symptom, and was regarded as part of the ataxia. There was no nystagmus. The sensibility, both superficial and deep, so far as could be determined by the usual clinical tests, was quite normal. Especially noteworthy was the preservation of the sense of posture and the stereognostic sense. In none of the cases was there a serious mental defect. The retardation was no more than might follow a severe disturbance of speech and incoördination and the restriction of opportunity imposed upon the child.

In none of the cases was there any tendency to grand mal or petit mal seizures. The convulsions occurring immediately after the birth had manifested no tendency to recur in later life.

Some improvement had taken place in all of the patients, and there were no indications of an increasing mental deterioration or progression of other symptoms. In this respect the ataxic form resembles the spastic type of Little with its well-recognized tendency to improvement.

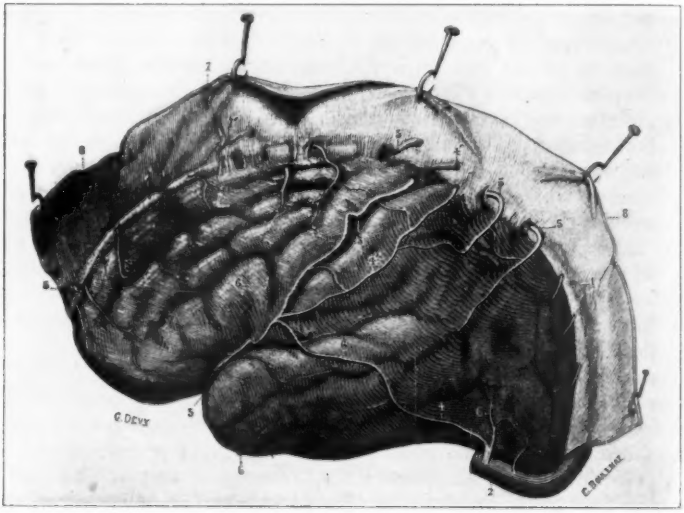
This very striking disturbance of motor incoördination I would refer to a disorder of the cortical functions, probably the result of a bilateral injury to the parietal area at birth. A lesion of this character would interfere with the development of the higher cortical centers engaged in the reception and elaboration of the memories of movements, and produce a *pure cortical ataxia;* or it might interrupt the commissural symptoms by which these memories are communicated to the motor area for the proper regulation of coördination, with a resulting *transcortical ataxia.* In either event a serious loss of the control and regulation of movement would of necessity result.

The primordial cortical centers which receive muscle and posture sensibility are not affected, for the general sensibility, both superficial and deep, is apparently preserved. It is probable therefore that the cortical centers subserving the function of common sensibility are either not included within the limits of the destructive lesion or, if they are injured, their function is subsequently restored by the compensatory activities of other centers, cortical and infracortical.

Pathological Considerations. The pathology of this group of cases in the absence of definite pathological evidence may only be surmised. I believe, however, that we are justified in the assumption that the lesions are of the same general character as have been found associated with other types of the cerebral birth palsies, differing only in their localization.

The pathological studies of Kundrat have shown that thrombosis of the superior longitudinal sinus with occlusion or rupture

of the cerebral veins is a by no means uncommon sequel of injury to the child's head during birth. This form of lesion is very probably favored, if not directly induced, by overlapping of the parietal bones during the process of molding of the fetal head. During the course of a prolonged and difficult labor, instrumental delivery, or precipitate parturition, the delicate cerebral veins may be torn from their connections as they traverse the intermeningeal space to enter the sinus, with consecutive hemorrhage over the surface of the brain, either subdural or intermeningeal.



Showing distribution of the cerebral veins on the external surface of the hemisphere. The dura mater is lifted to show the termination of the veins in the superior longitudinal sinus. *1*, superior longitudinal sinus; *2*, horizontal portion of lateral sinus; *3*, large anastomotic vein of Trolard; *4*, anastomotic vein of Labbé; 4^1, anastomosis between the vein of Trolard and the superior longitudinal sinus; *5*, *5*, *5*, ascending veins of the hemisphere; *6*, *6*, *6*, descending veins; *7*, branch of the middle meningeal anastomosing at 7^1 with an ascending cerebral vein as it enters the sinus; *8*, dura mater. (From Testut's Anatomy.)

The subdural type of hemorrhage is usually massive, consisting of a single large clot, and is commonly unilateral. Intermeningeal hemorrhages are more diffuse, often bilateral, and consist of a thin film-like clot of blood confined by the piarachnoid. This is greatest in the region of the superior longitudinal fissure where the cerebral veins cross to the sinus and gradually tapers off as it passes over the surface of the hemisphere toward the fissure of Sylvius. As the larger veins run their course in relation to the fissures there is a natural tendency for the blood to follow and gravitate in the depths of the larger cerebral sulci. Limitation of

the vascular lesion to the region of the fissure of Rolando would explain the occurrence of spastic diplegia, and a similar explanation might be offered for the manifestations of cerebral diataxia, namely, thrombosis or hemorrhage limited to the parietal veins of the cerebral cortex (see figure).

Kundrat also emphasized the importance of structural peculiarities of the margins of the parietal bones and intervening membrane to injuries of the sinus. He expressed the belief that thrombosis and hemorrhages of venous origin are much more likely to occur in infants with firm bony edges and narrow interosseous membranes, because of the mechanical interference with overlap in the process of molding of the fetal head. A narrow interstitial membrane and parietal bones, with thick heavy margins, would render the sinus liable to the dangers of compression, with consecutive thrombosis or hemorrhage of the cortical veins. Kundrat also performed the interesting experiment of attempting an injection of the cerebral veins through the exposed sinus while holding the parietal bones firmly in a position of overlap, and found that the backward flow of blood through the sinus was effectively blocked by the compressive action of the bones. The dangers of such a contingency in long-protracted labor are obvious notwithstanding the rich collateral circulation of the cerebral venous system.

The essential lesions of the cerebral birth palsy consist therefore of hemorrhage either subdural or intermeningeal, with more or less thrombotic softening of the cerebral cortex from obstruction to the cerebral venous system. These may occur in various combinations. In grave cases lacerations of the brain substance, ventricular and intracerebral apoplexy and hemorrhages at the base of the brain are occasionally observed. Involvement of the cortical venous system is, however, the most important from the clinical point of view, as the severer forms of injury are usually rapidly fatal and therefore have little more than a pathological interest.

Studies of the cerebral cortex in cases of sinus thrombosis also show that intermeningeal hemorrhage is by no means the most important factor in producing the serious consequences of birth palsy, but that grave and permanent changes in the cortex itself may result. Thrombosis of the cerebral veins produces retrograde engorgement of blood, with punctate hemorrhages in the corresponding vascular distribution, and is associated with edema, perivascular engorgement and degeneration of the ganglion cells. (Hunt.) Although a certain degree of collateral circulation may be established, the delicate cells and neurons of the superficial layers of the cerebral cortex would inevitably suffer considerable damage.

Any limitation of the venous thrombosis to a definite vascular distribution would play a very important role in the causation of

special clinical types and would explain the occurrence of purely spastic or purely ataxic types of this affection.

In the majority of cases the effusion or thrombosis takes place in the motor areas, producing the spastic type described by Little. That this lesion may be quite circumscribed is shown by the not infrequent occurrence of purely spastic states without mental defect, epilepsy, chorea or athetosis.

The ataxic type of cerebral birth palsy is, I believe, the result of a more or less circumscribed lesion of similar nature within the confines of the parietal area behind the fissure of Rolando. The anatomical distribution of the parietal veins of the cerebral cortex render such a lesion not improbable, the effusion of blood and cortical degenerations interfering with the proper development of the various centers and commissural systems which subserve the higher functions of the coördination of movement (see figures). A lesion of this character might affect a dissociation of the sensory and motor spheres of the cortex, producing a condition in which motion would be possible and the sensibility preserved, and yet the harmony and coördination of movement would be lost, a form of *transcortical ataxia* in which there is an inability to convey to the motor area the complicated memories of movements and their combinations, so essential for a precise coördinate act.

That the parietal lobes contain important centers for muscle sensibility and the memories of movements has been shown by a number of observers and is accepted by all authorities on cerebral localization. The parietal sensory area includes the postcentral convolution, the superior parietal lobule (PI) and the inferior parietal lobule (PII) and portions of the supramarginal and angular gyri. It includes as well the precuneus on the mesial surface of the hemisphere.

In this region are stored not only the sensations of individual joints and muscles, the proprioceptive sensibility proper, but also the higher and more complicated motor memories, the various acquired psychic memories of movements which are essential for the development of a finer and more delicate system of coördination.

Von Monokow has suggested that the *fasciculus centroparietalis*, an association bundle uniting the angular gyrus and the Rolandic area, may play a role in the cortical association of muscle sensibility. He regards the parietal convolutions PI and PII and their association tracts as of special importance for the representation of movements and combinations of movements. The sensations of the individual muscle groups and segments and tactile sensibility he would localize in the postcentral convolution.

It is now generally held that the simple perception of deep sensibility, including the muscle sense and the sense of posture, are localized in the ascending parietal convolutions (postcentral gyrus), probably in segmental relation to the corresponding motor

centers of the central convolutions (Mills), while the higher psychic perceptions of motor activity, namely, the memories of movements and combinations of movements are situated more posteriorly in the superior and inferior parietal lobule, a region which is concerned with the registration of higher motor memories.

Indeed, it is quite conceivable that the centers which are engaged in the perception of deep sensibility might escape or recover from the effects of a lesion, and yet the higher centers and commissural systems which register and transmit the more complicated motor memories be seriously impaired.

The view which has just been expressed as to the pathology and localization of the lesions in cases of cerebral birth palsy receives a certain confirmation from the clinical and pathological investigations of Holmes and Sargent of injuries to the superior longitudinal sinus from gunshot wounds of the head. In their large series of cases, comprising seventy observations, the conspicuous feature of the clinical picture was a bilateral spastic paralysis, with marked rigidity involving the legs, trunk and arms in the order named. Articulation and the movements of the face were usually not affected. The paralysis was greatest in the legs and gradualy diminished in an upward direction, thus corresponding to the arrangement of the motor centers in the Rolandic area. The accompanying muscular rigidity was extreme. By analogy one might say that such cases corresponded to the spastic type of Little's disease in which cortical hemorrhages and thrombosis of the longitudinal sinus and cerebral veins play an important role.

Sensory disturbances were also occasionally noted by Holmes and Sargent and their sensory investigations are of interest as bearing on the symptomatology of lesions of the parietal lobe. Their summary is as follows:

"The sensory disturbances are especially interesting, as they are almost always those of a pure cortical lesion unaccompanied by any shock effect. The appreciation of pain and temperature is unaffected, and there is no definite diminution of tactile sensibility, but a certain number of light contacts are not recognized; there is, however, no threshold alteration, and the proportion of those missed is not directly related to the intensity of the stimulus. On the other hand, the localization of tactile stimuli, the recognition of the position and of passive movements of the limbs, and of form, shape and size, as well as the discrimination of the compass points, may be seriously disturbed. The slightness of the affection of cutaneous sensibility has been frequently astonishing, as many patients have complained spontaneously of numbness and of having 'no feeling' in their legs." One of their cases was of special interest as indicating the possible course of events in the infantile ataxic group, the motor paralysis clearing up rapidly, with persistence of a high degree of ataxia.

It should be emphasized that these studies were made upon adults in whom cortical function was fully developed in contradistinction to the infantile group in which cortical centers and systems are still immature and in which several years must elapse before accurate sensory examinations are possible. This may in part explain the preservation of common sensation in the infantile group, with loss of the power of coördination.

In all other forms of ataxia, save the cerebellar ataxia, there is a disturbance of the deep sensibility, the degree of which usually determines the severity of the motor disorder. This is true of the peripheral, spinal, thalamic and cerebral ataxia.

Why should the deep sensibility be preserved and yet a severe degree of motor incoördination be present in the ataxic birth palsies? No definite answer can be given to this question at the present time which is not more or less theoretical in nature.

In the first place it is possible that the cortical representation for the perception of deep sensibility covers a larger field than is usually held—as is true according to von Monokow of the cortical motor zone, and thus a considerable area might escape injury. As the lesion occurs in early life there is also ample opportunity for compensatory replacements and substitutions on the part of subcortical centers and neighboring regions of the cerebral cortex. It is also possible that the sensory area for the perception of muscle sensibility is limited to the postcentral convolution and so might escape entirely the ravages of the lesion.

In the course of development of the cortical functions of motility the acquired memories of movements and their complex coördination are registered in a manner similar to the storing of the speech memories. These engrams represent a higher sensory elaboration of movement and would be all-important in the carrying out of precise and definite motor acts. If the region of the parietal cortex subserving this higher function of motility were injured on both sides in early life these memories would be improperly registered or the commissural fibers, through which they exert their influence on motility, would be interrupted with a resulting disharmony of movement. This would produce a cortical or transcortical ataxia, a disorder in the sphere of movement similar to apraxia or agnosia, in which the mechanism for the reception or transmission of higher motor memories would be seriously impaired, the bilaterality of the lesion preventing any compensatory activity on the part of the opposite hemisphere.

As the injury to the cortex takes place at birth, and as these cases cannot be subjected to accurate sensory tests much before the sixth or seventh year, there is ample opportunity for restoration and substitution of function. It is therefore not altogether surprising that the deep sensibility is preserved in conjunction with incoördination of the general motor activities. The cortical

centers subserving the function of protopathic sensibility either escape injury or are capable of being replaced by the compensatory activity of adjacent cortical areas.

The higher coördination activities of the muscular system are gradually acquired and are especially retarded and slow of development in this group of cases. That even these are susceptible of gradual development is shown by the clinical course of the cases.

Clinical Comments. The cerebral diataxia bears a certain resemblance to the cerebellar ataxia. The gait, like the cerebellar gait, may be somewhat titubating; there is dysdiadokokinesis and the speech has a dysarthric character not unlike that observed in cases of cerebellar disease. There is, however, no nystagmus, the disturbance of movement is not a dysmetria but a true ataxia, and the ataxia of the extremities is marked and persists in the recumbent posture. In the recumbent posture, with the extremities elevated and the eyes closed, there are ataxic movements, such as are observed in spinal ataxia and unlike the true cerebellar ataxia. Furthermore, the ataxia of the distal portion of the extremities is quite evident. There is also, especially during effort and mental excitement, a tendency to atactiform movements, of a more general character, which resemble or rather suggest a choreiform disturbance. They are not, however, spontaneous, but disappear in the relaxed and recumbent posture and appear only during muscular effort and movement. They are to be regarded as compensatory ataxic manifestations, similar to those occasionally observed in other forms of ataxia.

Another point of interest is the presence of a slight degree of hypotonia. It is well known that lesions of the parietal lobe may produce hypotonicity of the musculature, with diminution of the tendon reflexes. It is therefore not surprising to encounter a certain degree of hypotonicity in conjunction with cortical ataxia from lesions of the parietal lobe. In some of the cases described as cerebellar or atonic-astasic types of diplegia the nature of the ataxia and the associated hypotonia are somewhat suggestive of a parietal localization. This opens up a field for future investigation, and it is my belief that certain symptoms of diplegia which have heretofore been relegated to the cerebellum are really of cortical origin and referable to the higher cerebral centers for the control and coördination of movement.

In the above description I have attempted to outline the chief clinical features of the ataxic type of cerebral birth palsy or cerebral diataxia. It is the sensory equivalent of spastic diplegia, and like this affection shows a similar tendency toward improvement. In a pure form it is, however, comparatively rare.

Some of the cases probably recover or show a very considerable degree of improvement, and are then regarded as of cerebellar origin, an interpretation in which I was originally inclined to par-

ticipate. After a more detailed study of the motor phenomena, however, I feel convinced that the cortical mechanism is the essential structure involved.

The Ataxic Diplegia. This point of view finds a further confirmation in the not infrequent combination of ataxia and spastic paralysis in many cases of cerebral birth palsy, cases in which there are symptoms of generalized spasticity but modified by the association of ataxic symptoms. This constitutes a very grave form of the cerebral birth palsy, examples of which may be found in nearly every institution for the care of chronic diseases. The condition of motor weakness and spasticity being accentuated by the loss of the power of coördination.

Two such cases, aged ten and eleven years respectively, with the typical history of injury at birth, are at the present time under observation in my wards at the Montefiore Home and Hospital. Both cases are confined to the wheel chair and are unable to stand or walk. There is a generalized spastic state which is complicated by a profound general ataxia, any attempt to carry out a precise movement evoking a wild disorder of incoördination. There is no true chorea or athetosis, although the nature of the ataxic disorder suggests a choreiform disturbance.

The atactiform movements do not, however, occur spontaneously, but only on attempts at movement. This I believe is an important factor in differentiating the true chorea and athetosis.

Both patients are fairly intelligent and attend the hospital school. There are no epileptic manifestations. The speech is dysarthric, spastic and very difficult to understand. The sensibility, both superficial and deep, is normal. As in the pure ataxic type there is no demonstrable disorder of the sense of posture or the sense of movement. The stereognostic sense is preserved and any common object placed in the hand is promptly recognized. In these cases I believe there is a combination of lesions which involves the motor sphere causing general rigidity and also the parietal area with a resulting disturbance of coördination. The clinical picture is one of generalized spasticity and ataxia in combination, namely, a *diataxic diplegia.*

Many such cases are, I believe, regarded as choreiform or athetoid types of diplegia, the extreme atactiform disorder of movement simulating very closely chorea. If there is marked spasticity an athetoid tendency is suggested.

In a previous communication I presented the pathological evidence, indicating the relationship of chorea and athetosis to the caudate nucleus and putamen of the corpus striatum, and there can be no question as to the paramount importance of the corpus striatum in the production of true chorea and athetosis. While the relationship of the cerebral cortex to chorea and athetosis is

accepted by all systematic writers, the exact localization of this function is still one of the unsolved problems of brain pathology. In all such investigations the existence of a cortical diataxia should be carefully considered and its very close resemblance to such disorders as chorea and athetosis.

CONCLUDING REMARKS. A variety of clinical types of the cerebral palsies of childhood are now recognized, and as such are firmly established in medical literature. Important among these may be mentioned the various forms of the spastic type of Little, the atonic or flaccid type of Förster, and those of cerebellar origin.

In this paper I have directed attention to the existence of a pure ataxic type which may be distinguished from other forms as *diataxia cerebralis infantilis.*

The infantile cerebral diataxia is a special form of the cerebral birth palsy in which the symptomatology is characterized by a generalized disturbance of coördination without evidences of paralysis, spasticity, epilepsy or serious mental defect. The sensibility, both special and general, is apparently unaffected.

The clinical picture is believed to be dependent upon a bilateral vascular lesion in the parietal region, viz., hemorrhage and softening in the distribution of the parietal veins, resulting from an injury at birth.

The parietal area of the cerebral cortex is concerned with the reception and elaboration of higher motor memories, and a bilateral lesion in this region in early life would seriously interfere with the proper development of its cortical centers, association fibers and commissural systems. This would produce a disorder of the higher cortical functions of coördination—with a resulting *cortical* or *transcortical ataxia.*

The disturbance of coördination affects the speech, the gait and station and the use of the extremities, and is a pure ataxic disturbance.

It may be distinguished from cerebellar ataxia by the character of the motor disorder, its persistence in the recumbent posture and the absence of nystagmus. It may be regarded as the sensory equivalent of the spastic type of Little and is not infrequently encountered in combination with this affection, constituting a *diataxic diplegia.*

As in Little's disease, there is a tendency toward improvement, although the development of motility is always seriously retarded.

In its more severe forms this disorder of coördination may be associated with a tendency to atactiform movements which resemble superficially the phenomena of genuine chorea and athetosis; the recognition of which I believe to be of considerable importance in the eventual solution of the problem of the nature and localization of chorea and athetosis.

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ANGINA ABDOMINIS.[1]

By Edward H. Goodman, M.D.,

PHILADELPHIA, PA.

The term angina abdominis was introduced by Baccelli to describe painful seizures in the abdomen due to aneurysmal for-

[1] Read before the American Gastro-enterological Association.

mations in the vessels of the celiac axis, or due to arteriosclerosis of these same vessels. Another name for the same condition has been offered by Huchard, angina pectoris pseudogastralgique. Inasmuch as the name invented by Baccelli has been accepted and used by recent writers, notably Pal, Brunton and Williams, Minella, Breitmann and others, it is the term which should be used in the future for this disease.

Case History. The patient, a well-preserved woman, aged eighty-two years, apart from trivial illnesses of but short duration, has enjoyed good health for the greater part of her life. Twelve years ago she suffered with intense pain in the stomach, which was diagnosed at this time carcinoma ventriculi. In view of the present state of health the diagnosis is subject to question. Disregarding this attack, which resembled the seizure recently experienced, the patient rejoiced in the best of health and spirits until the early part of September, 1916. Following hard physical work connected with housecleaning, a task which no appeal to regard her years could make her depute to others, she was stricken with violent and intense pain in the umbilical region, which was accompanied by great anxiety, nervousness, restlessness and attempts at expulsion of gas per rectum and ex ore. These attacks having no relation to meals were repeated frequently, medication and diet proving unavailing, since September. She was seen by a surgeon in November, 1916, as gall-stones were suspected, but the diagnosis was not confirmed. Operation was disadvised, but no satisfactory conclusion was reached concerning the nature of the attacks. The surgeon's examination, which was most thorough, failed to reveal any organic lesion other than the arteriosclerosis.

The patient was counselled to remain in bed, to partake of nothing except milk, and was given a mixture of hydrochloric acid and pepsin, with occasional doses of sodium bicarbonate and nutmeg. For three days she was so much better that her physician permitted her to get out of bed, and again with utter disregard for her years, she cleaned a closet and laid unassisted a heavy carpet (November 23), being alone in the house at this time. Following this exertion she was seized with an excruciating pain in the umbilical region, with restlessness, extreme nervousness, attempts at expulsion of gas, no abdominal distention, no passage of urine or feces, some nausea, but no vomiting. The following morning this attack was duplicated in all characteristics except that the pain was less formidable. I saw the patient in company with the attending physician approximately about four hours after this second, and to date, last attack. The seizure on this day had been of short duration, as were all the others, that is to say, the acme of the frightful pain was sustained for only a few minutes, but intense abdominal soreness followed, and was present when I saw her, and lasted for a period of about twenty-four hours.

I found the patient (November 24) to be a remarkably well-preserved old lady, of frail build. The pulses were equal, regular, normal in rate, but the vessels were hard and easily palpable. The heart examination revealed a faint systolic murmur at the base, which was interpreted as being caused by aortic roughening.

The stomach was slightly dilated, there was a splash (milk had been ingested but a short time before) and some diastasis of the recti. The abdomen was very sensitive to pressure, particularly in the umbilical region, corresponding to a circular area about 6 inches in diameter, having the umbilicus as its center. There was marked throbbing of the abdominal aorta, no evidence of any enlargement of the vessels, and no murmurs. The reflexes were normal and the lower legs were slightly edematous. A rectal examination made by the surgeon five days previously, and being reported negative, was not repeated. The urine has contained albumin, but no sugar or casts. The blood-pressure was not taken at this time, as I had neglected to take my instrument, having been told it was essentially a stomach case. My neglect in this respect I have since regretted, as my request to the attending physician for a subsequent report of the blood-pressure was overlooked. However, four months later the pressure was found to be 130–60.

I believed we were confronted with an almost typical case of angina abdominis, and although skeptical as to the correctness of this view, the physician consented to give my therapeutic recommendations a trial. To the patient was explained what we believed to be the cause of her distressing attacks, and we gained her support in the view that physical overexertion or nervous strain, or both, were predisposing factors in calling an attack into being. Therefore the recommendation to rest during the day, and particularly a half-hour's rest on the right side, with a hot-water bottle to her abdomen following her meals, was readily agreed to. She was permitted to eat anything sparingly at the regular hours for meals and a glass of milk with a biscuit between meals. Only meat was restricted. Nitroglycerin, gr. $\frac{1}{100}$, was given every three hours, and potassium iodide, gr. x, three times a day. Pearls of amyl nitrite were left in the event of a recurrence of the pain.

Since November 24 the patient experienced the beginning of an attack, but the attack was cut short by the prompt use of amyl nitrite, and since that date there have been no further attacks. Potassium iodide and nitroglycerin have long since been discontinued; the patient is active in Red Cross work, and seems to be enjoying the best of health, the fear of cancer having, to her great relief, been permanently dispelled from her thoughts.

The symptoms as disclosed by the history of this patient seem almost typical of what has been described by others as angina abdominis, and a further discussion of the symptomatology of a classical case would seem unnecessary. Permit me to recall the

cardinal facts. The main feature of this woman's illness was the sudden agonizing pain which was directly associated with physical overexertion or nervous shock, thus in a way resembling the onset of angina pectoris. There is a prevalent notion that all attacks of angina pectoris are determined by emotional or physical effort, yet from Heberden himself we learn that attacks may occur during sleep and often during rest. Diderot records an attack which aroused him from sleep (Allbutt). Nevertheless, many cases of angina pectoris are precipitated by exertion, and in this patient of mine effort seems to have been the exciting factor. The pain is described by the patient as being horribly severe, and after the first seizure the dread of another and the realization that others were to occur, since the attacks were repeated, made her life one of continuous apprehension.

Accompanying the pain there were efforts at expulsion of gas by mouth and rectum, and when belching could be effected there seems to have been some slight relief. The bowels were slightly constipated. There was slight nausea but no vomiting.

The situation of the pain was about the umbilical region and radiated to the back, and following the seizure there was great sensitiveness on slight pressure about the umbilical region for an area 6 inches in diameter.

The diagnosis should not be difficult, and to my mind the main support of the correct diagnosis should be based on the frightfulness of the pain, the short duration of the same, the occurrence of the pain in arteriosclerotic individuals, usually advanced in years, in whom, apart from the arteriosclerosis, there can be found no appreciable sign of disease. Men are said to be more frequently affected than women.

The differential diagnosis must, in some cases, be made between abdominal angina and other diseases associated with gastric crises. Here it may be stated that Allbutt believes that epigastric angina is but a somewhat aberrant form of ordinary angina, and due, broadly speaking, to disease of some part of the thoracic aorta; but in angina "more definitely abdominal," he thinks it may be due to disease of the lower portion of the aorta or in other large vessels there. Osler is disposed to discredit the diagnosis, regarding abdominal angina as but the abdominal symptoms of angina pectoris. In this patient of mine, despite her years, no symptom of any cardiac disease had been complained of. There were no transient seizures without pain in which "there was a general suspension of the minor operations of nature for three or four seconds," called angina sina dolore.

Abdominal aneurysm should not be a matter of much moment in the differential diagnosis, as the pain is never anginoid, but rather boring in character, and situate in the left abdominal region. (Of 179 cases reported by Nixon none had anginoid pains.)

Tabes dorsalis should be readily distinguished, as the reflexes point the way to the true diagnosis, unless, unhappily, the patient is a sufferer from both tabes and from angina abdominis. The pain in tabes is intercostal rather than abdominal.

Closure of the mesenteric vessels I mention as an improbable differential diagnostic difficulty, on account of its extreme infrequency and because of the impossibility of recognizing the condition, unless one has been the observer of a previous case. Closure of the mesenteric vessels may take place in either the veins or arteries as a result of thrombosis or embolism. For thrombosis, arteriosclerosis itself is an underlying predisposing cause; for embolism valvulitis is the contributing factor. The onset of mesenteric closure is sudden and leads rapidly with great pain to collapse. Bloody vomitus is seen, and the stools, which contain blood, may be either obstipated or diarrheic. The abdomen becomes distended, painful and resistant to touch, with dulness in the dependent portions of the abdomen. The temperature is normal, pulse is rapid and soft and the patient gives the impression of one suffering with severe peritonitis or intestinal obstruction. In no case should abdominal angina be mistaken for mesenteric closure.

I should prefer to leave the question of pathology undiscussed, as but few cases have been studied satisfactorily from the clinical and pathological sides. Various suppositions have been suggested, but "clinical and pathological precision has yet to contribute to a better analysis of these phenomena." (Allbutt.)

So far as therapy is concerned, rest is an important measure, and avoidance of physical or nervous strain is to be recommended. Diet is to be that generally recommended for individuals of advanced years. Massage and faradization of the abdomen have been advised, and the homely hot-water bottle is not to be despised. Potassium iodide, sodium nitrite, sodiotheobromin, salicylate, nitroglycerin, amyl nitrite may each have its place, alone or in careful combination. During an attack if amyl nitrite fails, morphin, hypodermically, may be employed.

THE RELATION OF LUPUS ERYTHEMATOSUS DISCOIDES TO TUBERCULOUS INFECTION.

By Richard S. Weiss, M.D.,

and

J. J. Singer, M.D.,

DEPARTMENT OF INTERNAL MEDICINE, WASHINGTON UNIVERSITY MEDICAL SCHOOL, CLINICS OF TUBERCULOSIS AND DERMATOLOGY, ST. LOUIS, MISSOURI.

The etiology of lupus erythematosus has been one of the obscure problems of dermatology ever since the first description of the dis-

ease by Cazenave[1] in 1851. Investigations up to about the year 1910 have all tended to show that the disease is in some way related to tuberculosis. At first the belief seemed to be that it was a true inoculated tuberculosis of the skin. (Riel and Paltauf;[2] Klotz.[3]) Shortly after this, however, the tubercle bacillus toxin theory was elaborated and, led by certain French dermatologists (Hallopeau,[4] Boeck,[5 6] and Besnier), this theory seems to have been accepted by most observers as the probable etiological explanation.

In recent years doubt has been cast upon this theory. Unna,[5] Radcliffe Crocker,[5] Pick,[7] Sequeira and Balean,[8] Galloway,[9] Warde,[10 11] Pusey,[12] Engman and Mook[13] are all on record against it, although many of them and other observers believe that it is of toxic origin and should be classed with the polymorphic erythema group of skin diseases.

The basis for the tubercle toxin theory was the frequent finding of evidence of tuberculosis both clinically and postmortem. It is unnecessary here to go into the literature and review these statistics. Suffice it to say that in a large proportion of cases of this disease it has been observed that (*a*) there is a family history of tuberculosis; (*b*) there has been a positive reaction to the tuberculin test; (*c*) that there is clinical evidence of tuberculosis; (*d*) that evidence of tuberculosis was found postmortem. We shall take up these points and discuss them later.

We have studied 12 cases of lupus erythematosus discoides from the skin and tuberculosis clinics of the Washington University Dispensary. These cases were approached from as many angles as possible; a detailed history, with special reference to past and present illness and previous exposure; a careful physical examination together with roentgen plates; tuberculin tests; Wassermann reaction; and the pulse, temperature, and weight record. A brief synopsis of each case follows:

CASE I.—A. H., white; female; aged fifty years. C 1. No. 27846.

Family History. Negative.

Personal History. Erysipelas in 1889. Since then has never had any serious illness.

Wassermann Reaction. Negative.

Tuberculin Test. Positive; reacts to $\frac{1}{2}$ mgm. O. T. but not to $\frac{1}{10}$.

Skin disease began at the age of forty-five years. Presents lupus erythematosus of the lips.

Physical Examination of the Chest. No evidence of tuberculosis found. Roentgen-ray plate; air content good, somewhat better in the lower right lobe than elsewhere. Right lung: arborization is distinct from rather dense hilus, with several small dense glands. Diaphragm normal. Left lung: similar to right.

Diagnosis. Healed tuberculosis of childhood. (Diagnosis made on roentgen plate.)

CASE II.—K. A., white; female; aged thirty-one years. C 1. No. 27842.

Family History. Negative.

Personal History. Never had any serious illness.

Wassermann Reaction. Not done.

Tuberculin Test. Positive; marked local reaction to $\frac{1}{10}$ mgm. O. T.

Skin disease begun at the age of thirty years. Presents single patch of lupus erythematosus on side of nose.

Physical Examination. Harsh breathing, anteriorly and posteriorly in both lungs, but no increase in whisper; probably "cold."

Roentgen Plate. Fairly good air content in both lungs. Right lung: upper lobe shows thickening of interlobular lines, but no marked approximation. The hilus is fairly dense, but no definite nodules are seen. Extension to the lower lobe dense at the proximal area, but not in the axillary area. Diaphragm held at an angle of 90 degrees. Left lung shows thickening of the interlobular lines; no definite shadow. Hilus is fairly dense. Lower lobe markings faint, with diaphragm flattened. Diagnosis: Chronic tuberculosis; chronic pleurisy. No clinical tuberculosis.

Diagnosis. By roentgen rays shows evidence of an old chronic tuberculosis.

CASE III.—L. R., white; female; aged thirty-three years. C 1. No. 22585.

Family History. Grandmother and one cousin died of tuberculosis; otherwise negative.

Personal History. Measles, scarlet fever, mumps, pertussis, and varicella in childhood. Was very delicate as an infant. Following scarlet fever and measles, at the age of five years, was very "nervous" for a number of years. Malaria (?) at fifteen. Nervous prostration from sixteen to twenty. Smallpox at twenty-one. Typhoid at twenty-five, of moderate severity, followed by nervousness. Mild attacks of grip, bronchitis, and tonsillitis since then.

Wassermann Reaction. Negative.

Tuberculin Test. Von Pirquet positive in 1914. 1916 reacted to $\frac{1}{10}$ mgm. O. T. and focal reaction in lesions.

Skin disease began at the age of twenty-nine years. Presents lupus erythematosus of face. Scars of old lesions on scalp, backs of fingers, and arms.

Physical Examination. Deficient fat. Slight muscle spasm in upper thorax on both sides. Breathing in left apex distinctly harsher than in right. Breathing in right apex seems to be diminished, but owing to the patient being nervous she does not breathe deeply. Whisper is normal. Slightly impaired resonance posteriorly. Roentgen plate. Fair air content in both upper lobes. Left lung: interlobular markings not approximated. Hilus not dense. Extension from hilus is dense, but not closely approxi-

mated. Right lung is similar to left except that the diaphragm is more regular.

Diagnosis. Early pulmonary tuberculosis. (Diagnosis made on history and physical examination. Roentgen plate shows nothing definite.)

CASE IV.—F. P., colored; female; aged thirty-six years. C 1. No. 32335.

Family History. One sister died of pulmonary tuberculosis; otherwise negative.

Personal History. Patient states that she had "scrofula" in childhood, but can give no details. Has had no serious illness since.

Wassermann Reaction. Not done.

Tuberculin Test. Not done.

Disease began at the age of thirty-five years. Presents lupus erythematosus generally distributed over the face and under and on the ears.

Physical Examination. Impaired resonance over the right lung anteriorly, together with increased whisper both anteriorly and posteriorly. Diminished breathing on the right side posteriorly. No history of loss of weight, fever, or sputum.

Roentgen Plate. Not obtained (patient only made two visits to clinic). Diagnosis without roentgen rays is suspected tuberculosis.

CASE V.—E. F., white; female; aged forty-three years. C 1. No. 861.

Family History. Negative.

Personal History. Has no bearing on the case.

Wassermann Reaction. Negative.

Tuberculin Test. Positive; von Pirquet.

Disease began at about the age of twenty-six years. Presents lupus erythematosus. An extremely severe case of many years' duration, involving the face, ears, and neck in erythematous and scaly plaques, with much scarring and atrophy. Chilblain circulation.

Physical Examination. Expansion poor. No marked dulness, but some diminished resonance posteriorly. Diminished breathing in both lungs, both anteriorly and posteriorly. Whisper slightly increased, lower right anteriorly and upper left posteriorly. (Obesity of patient may account for diminished breathing.)

Roentgen Plate. Air content fairly good in both lungs. Left upper lobe between second and fourth ribs somewhat hazy. Hilus rather dense. Diaphragm normal. Right lung: hilus much denser than left. Several nodules seen. Some approximation of interlobular lines, especially of middle and lower lobes. Plate not taken in deep inspiration.

Diagnosis. Healed tuberculosis of childhood. No definite adult tuberculosis. (Patient received tuberculin treatment up to 20 mgm., with no effect upon the progress of the disease, but a focal reaction in the skin lesions was observed.)

CASE VI.—E. B., white; female; aged thirty-three years. C 1. No. 15223.

Family History. Negative.

Personal History. Never had any serious illness.

Wassermann Reaction. Not done.

Tuberculin Test. Positive.

Disease began at the age of thirty years. Presents lupus erythematosus. Several small erythematous and scaly plaques on the sides of the nose and adjoining cheeks.

Physical Examination. No infiltration demonstrable by percussion. Slight muscle spasm anteriorly and posteriorly of right thorax. Whisper sound increased posteriorly in right upper lobe. Few moist rales in left lung anteriorly and an occasional rale in right lung posteriorly. Roentgen plate. Air content in both lungs fair. There is an approximation of the interlobular lines in both upper lobes, but no thickening. The hilus on both sides is dense. The costal angles are deep. Plate not taken in deep inspiration. Temperature showed an occasional rise to 99+, and with the physical examination make diagnosis of pulmonary tuberculosis.

CASE VII.—F. S., white; male; aged thirty-four years. C 1. No. 18058.

Family History. Negative.

Personal History. Never had any serious illness.

Wassermann Reaction. Not done.

Tuberculin Test. Not done.

Disease began at about the age of twenty-five years. Lupus erythematosus. Scaly, erythematous plaques on nose and cheeks.

Physical Examination. Breath sounds are distant and diminished in both upper lobes. Whisper is increased in the right lung, more marked in the upper lobe posteriorly. Muscle spasm in posterior right thorax. Expiration prolonged and harsh posteriorly.

Roentgen Plate. Shows good air-containing lungs. The hilus on both sides is dense, with fairly dense extension. Both apices clear. Interlobular markings are distinct and not dense. Diaphragm normal.

Diagnosis. Chronic hilus tuberculosis (?). (Diagnosis made by roentgen plate and physical examination.)

CASE VIII.—L. K., white; female; aged forty years. C 1. No. 32106.

Family History. Negative.

Personal History. Never seriously sick.

Wassermann Reaction. Negative.

Disease began at the age of thirty-nine years. Lupus erythematosus. Small erythematous, scaly patch on side of nose.

Physical Examination. Harsh breathing over the bases of both lungs posteriorly, with slight increase of whisper. No definite signs of tuberculosis.

Roentgen Plate. Air content of both lungs good. Right lung: bronchial branches well marked and can be traced to periphery. Hilus very dense, with extension to lower lobes slightly thickened. Several large dense glands are seen. Diaphragm negative. Costophrenic angle deep. Left lung: apex slightly hazy as compared to right. Otherwise findings are similar.

Diagnosis. Healed tuberculosis of childhood. No adult tuberculosis.

CASE IX.—E. B., white; female; aged forty-five years. C 1. No. 28632.

Family History. Negative.

Personal History. No bearing on case.

Wassermann Reaction. Negative.

Tuberculin Test. Not done.

Sputum Examination. Negative for tubercle bacilli.

Skin disease began at the age of forty-four years. Lupus erythematosus. Plaques scattered over the front and side of the face.

Physical Examination. Breath sounds diminished in both upper lobes. Few shifting, dry, and moist rales in both upper lobes. Impaired resonance in both upper lobes, anteriorly and posteriorly; not marked, however. Lower lobes normal.

Roentgen Plate. Air content fair, less in upper lobes. Right lung: Upper lobe, arborization thickened and interlobular lines are more or less approximated in axillary and mammary lines. Hilus dense, with many dense nodules. The extension to lower lobe is thickened and somewhat interlaced, with small pinhead-size points of density. Diaphragm normal. Costal angle deep. Left lung: Rather dense shadow in the upper lobe and marked thickening of the interlobular lines. A large nodule, size of a dime, is seen. Air content good in the lower lobes. Diaphragm irregular.

Roentgen Plate. Together with the physical examination and a constant temperature of 99° to 100° make the diagnosis of chronic pulmonary tuberculosis. Active tuberculosis of both upper lobes.

CASE X.—C. J., white; female; aged twenty-seven years. C 1. No. 21814.

Family History. One uncle by marriage died of pulmonary tuberculosis. Otherwise negative.

Personal History. Typhoid at twelve years. Frequent attacks of tonsillitis in childhood. Pus tubes and operation at age of nineteen, followed by "stomach trouble," which lasted on and off about six years. No history of syphilis.

Wassermann Reaction. Positive 4+.

Tuberculin Test. Not done.

Disease began at the age of twenty-four years. Presents lupus erythematosus. Extensive butterfly distribution.

Physical Examination. Right lung: anteriorly, breath sounds somewhat diminished except for an area in the second intercostal

space, near the sternum, where the breath sounds are very harsh and there is increased whisper. Lower lobe normal. Left lung: Upper lobe, anteriorly, breathing is diminished, but the whisper is loud here and there. Lower lobe is normal. Posteriorly, right lung, impaired resonance, with diminished breathing, no increased whisper. Left lung: breath sounds somewhat harsh, with increased whisper and impaired resonance. Lower lobes negative.

Roentgen Plate. Air content good in lower lobes, only fair in upper lobes. Left lung: Upper lobe: approximation of interlobular lines but not very dense. Many small dense nodules in this area. Hilus is broad and dense with several dense nodules. Lower lobe: bronchial arborization is not dense or interlaced. Diaphragm somewhat irregular. Lung dips deeply into costophrenic angle. Right lung, upper lobe, interlobular markings are dense but not as closely approximated as the left. Many nodules seen in this area. Hilus very dense and broad; many nodules seen. Middle lobe: arborization is distinct to periphery and there is a moderate amount of approximation. Lower lobe: arborization is dense and interlaced, and there is more or less approximation of interlobular lines. The proximal third of the diaphragm is held up by adhesions. Lung dips deeply into costophrenic angle.

Diagnosis. Chronic pulmonary tuberculosis. (Note: As the physical signs are not classical for tuberculosis and the Wassermann reaction is 4+ there is some doubt as to whether the lung condition is tuberculous or luetic.)

CASE XI.—S. W., white; female; aged thirty-one years. C 1. No. 19292.

Family History. One aunt and two uncles died of pulmonary tuberculosis. Otherwise negative.

Personal History. Usual diseases of childhood. Generally good health until the age of twenty-four years, then had an attack of "inflammatory rheumatism," affecting the finger-joints only. At the same time had phlebitis in the right leg. The rheumatism in the fingers would get better and worse, and lasted about three years. The phlebitis kept the patient in bed about ten weeks and then got entirely well. About one year after this, phlebitis started in the left leg and kept the patient in bed about six weeks. Three weeks later the patient had a second attack of diphtheria. At the age of twenty-eight years she had two ulcers, one on each ankle, which eventually healed. No other serious illness. No menstrual disturbance.

Wassermann Reaction. Negative.

Tuberculin Test. Positive. Reacted to $\frac{5}{10}$ mgm. O. T.

Skin disease began at the age of twenty-nine years. Presents lupus erythematosus. Plaques all over the face and ears.

Physical Examination. No definite signs of tuberculosis.

Roentgen Plate. Considerable air content. Left more than right. Right lung: there is no approximation of the interlobular lines of the upper lobes; a few small dense nodules are seen. The hilus is fairly dense and shows numerous nodules varying in size from a pinhead to a pea. Lower lobe: a few nodules are seen. The diaphragm is normal. Costal angle is deep. Left lung: interlobular markings are not distinct; no approximation. The hilus is less dense than the right. Lower lobe: the interlobular markings are distinct almost to periphery. Diaphragm is normal. The costal angle deep.

Diagnosis. Healed pulmonary tuberculosis of childhood. (No clinical tuberculosis.)

CASE XII.—N. D., white; female; aged fifty-two years. C 1. No. 18374.

Family History. As the patient remembers it one or two cousins had tuberculosis. Otherwise negative.

Personal History. Smallpox at eighteen months. Measles at five. Remittent (?) fever at fifteen. Both legs severely frozen at eighteen. Chronic rheumatism at thirty-seven; affected the knees and ankles and lasted one year. Ever since then gets slight attacks on occasion. Had a fatty tumor removed from the neck at the age of twenty-five. No history of lues. Never has been pregnant. Separated from husband for past twenty years.

Wassermann Reaction. Not done.

Tuberculin Test. Negative.

Skin disease began at the age of thirty-two. Present lupus erythematosus, butterfly distribution on face and a few lesions on scalp.

Physical Examination. Breath sounds in the right lung anteriorly are rather harsh, with no increased whisper. Left lung: upper lobe, breathing diminished, whisper not increased. Lower lobe: same findings. Posteriorly, similar findings. Chest is hyperresonant throughout.

Roentgen Plate. Shows a rather "brilliant" condition of both lungs, with the left extending farther down than the right. Left lung: hilus fairly dense, with several dense nodules. The interlobular markings are distinct to periphery, but are not approximated. There is a hazy appearance in the upper lobe near the axillary line. Diaphragm forms an angle of 90 degrees, the lung dipping down into the costal angle. Right lung: apex is cloudy. No interlobular lines seen. Hilus is quite dense, with numerous nodules. The interlobular markings are distinct to periphery, but not approximated. The diaphragm makes an angle of 90 degrees and is lower than the left. Lung dips deeply into the costal angle.

Diagnosis. Healed tuberculosis of childhood. Clinical diagnosis: chronic bronchitis; emphysema; chronic pleurisy (fibrinous).

We are therefore able to present 12 cases of lupus erythematosus

of the discoid type, 10 of which showed evidence of tuberculosis; 4 chronic tuberculosis; 1 active tuberculosis, and 5 healed tuberculosis of childhood. (We believe that the other 2 cases are tuberculous, but cannot present good proof.)

Let us compare these results with the results of others.

Boeck[6] investigated 36 cases and found pronounced symptoms of tuberculosis in 83 per cent. of them and a family history of tuberculosis in one-half of the balance of the cases. At a meeting of the Section on Dermatology of the British Medical Association[5] he gives the following figures: of 42 common, discoid, adult cases 28 presented indubitable evidence of tuberculosis, past or present, and 8 presented strumous ophthalmia: that is, 86 per cent. showed evidence of tuberculosis in some form.

Roth[14] collected 250 cases from the literature, and in 70 per cent. there was evidence, more or less pronounced, of tuberculosis.

Pick[7] reports that 18 of 43 cases of the discoid type showed evidence of tuberculosis (42 per cent.).

Sequeira and Balean[8] state that they found evidence of tuberculosis in 18 per cent. of their discoid cases and a family history of tuberculosis is not less than 80 per cent.

Kopp[15] found evidence of tuberculosis in 18 out of 38 cases (45 per cent.).

Gunsett[6] did 20 autopsies on cases of lupus erythematosus, and in only 9 was there evidence of tuberculosis.

A table was prepared for us of all the cases of lupus erythematosus of the discoid type appearing in the *British Journal of Dermatology* and in the *Journal of Cutaneous Diseases*. These cases numbered 225 in all. Tuberculosis was mentioned as being present or absent in only 64 of them. Of these 64 cases, 18, or 28 per cent., showed evidence of tuberculosis and 46, or 72 per cent., did not. In 69 cases mention was made of family history of tuberculosis; 28, or 40 per cent., were positive and 41, or 60 per cent., were negative. The tuberculin test was mentioned in only 9 cases, and of these 7 were positive and 2 negative. (These statistics were prepared by W. Kenneth Brown, of the senior class, Washington University Medical School.)

The statistics presented by Boeck and others led them to the belief that there was a direct connection or relation between lupus erythematosus and tuberculosis. Our statistics tally with many of theirs and, following out their line of thought, we might present the same conclusions; but our deductions, based on the modern conception of tuberculosis, are decidedly different.

We shall now take up in detail the evidence presented in the past, comparing it with ours and discussing its value.

First, all the evidence that has been presented is purely presumptive. We have been unable to find records of any work that shows a direct connection between the two conditions.

FAMILY HISTORY OF TUBERCULOSIS. Because quite a large percentage of cases of lupus erythematosus have a family history of tuberculosis it is presumed that this fact lends weight to the theory that there is a connection between the two diseases. Sequeira and Balean (*loc. cit.*) state that there is a family history of tuberculosis is not less than 80 per cent. of their cases. This is very high, and certainly might lend weight to the presumption were it not for other facts that must be taken into consideration. Boeck[6] finds a family history in only 8.8 per cent. of his cases. In our series 41.7 per cent. had a family history of tuberculosis. If we average these perhaps we shall come somewhere near the truth with 43.5 per cent. We are of the opinion that with careful questioning of patients fully as large a percentage could be found in many other conditions, say acne for instance. Then we would have to suspect that there was a relation between acne and tuberculosis.

TUBERCULIN TEST. "It is always found that between 40 and 60 per cent. of humanity react to the subcutaneous tuberculin test, provided that it be repeated with ascending doses three or four times" (Fishberg[17]). A positive tuberculin test indicates tuberculous infection and not necessarily tuberculous disease in a clinical sense (Hamman and Wolman[18]). Tuberculin has no effect on uninfected individuals (Hamburger[19]), but practically there are no uninfected individuals. We believe that Fishberg's statement can be modified to this effect, that practically all individuals above the age of twenty and living in large cities react to the tuberculin test. One of us (Singer) has been trying out the intradermic tuberculin test. In 49 out of 50 cases this test was positive; and here again we emphasize that the positive tuberculin test means tuberculous infection and not necessarily tuberculous disease. Our statistics, combined with those cases gathered from the *British Journal of Dermatology* and the *Journal of Cutaneous Diseases*, give 81 per cent. positive (13 out of 16 cases). With the intradermic test we have reason to believe that they all would be positive. A positive tuberculin test in cases of lupus erythematosus lends practically no weight to the theory that there is a relation between the disease and tuberculosis.

In this connection it would be well to discuss these facts; a focal reaction has occasionally been reported in the lesions of lupus erythematosus following the injection of tuberculin, and it is on record that lupus erythematosis has improved or been cured by the tuberculin treatment. The recent work of Engman and McGarry,[20] who caused the disappearance of the disease by the intravenous injection of foreign protein (typhoid and colon vaccine), shows that this is a foreign protein reaction and should not be interpreted as evidence of the relationship of the two diseases. Several of the cases which they report are included in our series, and we mention in particular their Case No. II, which is our Case No. V.

Here a focal reaction was observed in the lesions upon the injection of tuberculin. This case, as well as their other cases, cleared up wonderfully upon the intravenous injection of typhoid vaccine, *i. e.*, foreign protein. However, should we later on have to revise our conception of foreign protein reaction, these cases would still be open to question. They may easily have been lupus erythematoide of the Leloir type or lupus erythemato-tuberculeux of Besnier or a mixed type of lupus vulgaris and lupus erythematosus (Kyrle[21] and Spitzer[22]).

Clinical Evidence of Tuberculosis. With modern methods of diagnosis, clinical evidence of tuberculosis can be discovered in an extremely large number of individuals. We venture to say, with little fear of contradiction, that fully 90 per cent. of all individuals above the age of twenty and living in cities are subjects of tuberculous infection or tuberculous disease. Tuberculous infection in early life is the rule and not the exception, and so by far the great majority of us are "vaccinated in childhood against tuberculosis." (E. L. Opie.) In our series of cases 83 per cent. showed evidence of tuberculosis, and we have reason to believe that this figure should be 100 per cent. But of the 10 cases showing indisputable evidence of tuberculosis, 5 showed healed tuberculosis of childhood, and certainly are not tuberculous in the sense that they were suffering from consumption; 4 cases showed chronic tuberculosis and also were not tuberculous in the sense that they were suffering from consumption. Only 1 case presented active tuberculosis. Healed tuberculosis of childhood means calcified nodules in the lungs or mediastina. Chronic tuberculosis means a walled off, fibroid condition. Therefore we make the point that in 9 out of 10 of our cases it is very improbable that a circulating tubercle bacillus toxin is responsible for the skin condition.

Postmortem Evidence of Tuberculosis. Kopp (*loc. cit.*) found evidence of tuberculosis postmortem in 18 out of 38 cases of lupus erythematosis. He does not regard his results as evidence of the tuberculous origin of the disease. We arrive at the same conclusion but by different reasoning.

From the time of Laennec (1831) up to the present more and more evidence of the ubiquity of tuberculous infection, as demonstrated postmortem, has been accumulating. Practically every succeeding investigator has found a higher and higher percentage of tuberculous infection and disease on his postmortem table. We shall refer only to the latest of these reports. (For complete discussion of this subject see Fishberg, *loc. cit.*, p. 33 *et seq.*)

E. L. Opie,[23] by a most ingenious method of postmortem investigation (roentgen-ray pictures of the inflated and extirpated lung followed by minute section and search with the skiagram as a guide), has had the following results: Of 50 patients dying of diseases other than tuberculosis, each and every one showed lesions of tuber-

culous infection or tuberculous disease. These cases were from all the services of the Barnes Hospital, and they furnish convincing proof of the ubiquity of tuberculous infection and tuberculous disease. The finding of tuberculosis postmortem, in our opinion, is not valid evidence of a relation between lupus erythematosus and tuberculosis.

The facts then are these: Fully 90 per cent. of adult humanity living in large industrial centers are the subjects of tuberculous infection or tuberculous disease. Moreover, practically no cases of lupus erythematosus are observed among the inmates of large tuberculosis sanitariums, and the histopathology of lupus erythematosus bears no resemblance to any known form of tuberculosis. We therefore maintain that there is no evidence of an etiological relation between lupus erythematosus discoides and tuberculosis. The rarity of the one and the ubiquity of the other, to say the least, argue strongly against any such relationship.

SUMMARY. Twelve cases of lupus erythematosus discoides were carefully examined for tuberculosis. All available diagnostic methods were employed and a conclusion was reached in each case by study of the combined results and not from any single diagnostic proceedure.

Ten out of twelve of these cases showed indubitable evidence of tuberculosis, past or present, and it is believed that the other two are also tuberculous.

In only one of our series of cases that were definitely tuberculous was there any likelihood of the presence of a circulating tubercle bacillus toxin.

The evidence presented in the past to prove that lupus erythematosus discoides bears a relation to tuberculosis is purely presumptive.

Critical analysis of this evidence shows that conclusions drawn from it are based upon erroneous deductions.

CONCLUSION. In our opinion no evidence has been presented as yet that shows a relation between lupus erythematosus discoides and tuberculous infection or tuberculous disease.

It has been shown that tuberculosis past or present may be demonstrated in practically all cases of lupus erythematosus discoides, and this fact should only be interpreted as further evidence of the ubiquity of tuberculous infection and not as evidence of an etiological relationship between the two diseases.

We desire to thank Drs. George Dock and M. F. Engman for permission to use the cases reported and for their helpful suggestions in preparing this study.

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ANEURYSM OF THE ABDOMINAL AORTA WITH RUPTURE INTO THE DUODENUM.

By S. B. Marlow, M.D.,

and

F. H. Doubler, M.D.,

Boston, Mass.

(From the Medical and Pathological Services of the Peter Bent Brigham Hospital.)

That abdominal aneurysm is an infrequent condition both clinically and at autopsy is well known. Among 16,000 admissions to his wards at Johns Hopkins, Osler found only 16 cases; in Vienna 3 cases were found in 19,300 autopsies; Bryant has described 54 cases in 18,678 autopsies at Guy's Hospital; 13 cases in 6974 autopsies were found in the postmortem records of the Bristol Royal Infirmary.

Nixon, in 1911, collected and tabulated 233 cases of abdominal aneurysm including the above. Death resulted from rupture in 152 of these cases. In 149 the rupture took place either retroperitoneally or intraperitoneally; in 3 cases the rupture occurred into the gastro-intestinal tract; one of these, an aneurysm of the splenic artery, burst into the colon; the second is recorded as resulting in death from sudden collapse and profuse rectal hemorrhage. At autopsy cirrhosis of the liver was found, no mention being made as to the condition of the aneurysm, so that it is ques-

tionable whether rupture of the aneurysm or of a varix was the cause of death. The third case, a saccular aneurysm along the celiac axis and small aneurysm of the thoracic aorta, ruptured into the first part of the duodenum.

Since the publication of Nixon's tabulated series in 1911 there have occurred in the literature 11 other cases. Two of these cases ruptured into the gastro-intestinal tract. One case, reported by S. M. Zypkin, ruptured through a perforating ulcer into the stomach, so that at autopsy this organ was found filled with blood clot. The second case reported by E. A. Tozer ruptured into the duodenum. It was a saccular aneurysm of the abdominal aorta situated below the origin of the superior mesenteric artery. Tuberculosis of the aorta was found to be extensive and the etiological factor. Thus from a total of 244 cases 5 cases of abdominal aneurysm have occurred, with rupture into the gastro-intestinal tract.

The following are two cases, remarkably similar, of abdominal aneurysm which ruptured into the duodenum.

CASE I.—L. M., male, aged thirty-nine years, entered the Peter Bent Brigham Hospital June 25, 1916, complaining of "ulcer of the stomach."

Family History. Negative. Married sixteen years. Wife and two children well. One miscarriage. Habits are good; occupation hygienic.

Past History. Measles in childhood. Rheumatic fever fifteen years ago. He has had night-sweats for the past few weeks. There has been a loss of 20 pounds in weight in the last five months the patient thinks.

Present Illness. Started about January, 1916, with an indefinite sense of weakness. At that time the patient was worried over business affairs. There were "vague" symptoms which he could "not lay his fingers on." These persisted, and he thinks he gradually lost weight and strength. Previous to May 31, 1916, there is no history whatever of abnormal gastric or abdominal symptoms. On that day, while boarding a street car, he was taken with cramp-like pains "in the stomach." He walked into the car, sat down, fainted, and was unconscious for "ten minutes." When he came to he was covered with a drenching sweat. He proceeded home, arriving about one and a quarter hours later, and about ten minutes thereafter vomited fresh blood three times. The patient thinks that he vomited a considerable quantity, but has no idea as to the amount. He has since been on a restricted diet, and has been in bed practically all the time. There have been five subsequent spells of vomiting fresh blood, the last occurring eleven days ago. The patient has grown progressively weaker and more exhausted. His wife has noticed that most of the stools in the past four weeks have been tarry.

Physical Examination. The patient was a man, aged thirty-nine years, of medium stature, emaciated. On admission his gait was staggering, evidently due to weakness. Examination of his head, neck, lungs, heart, extremities, and reflexes was unimportant. His pulse was good. There was no thickening of any of his peripheral arteries. His heart was somewhat irregular. Examination of his abdomen showed distinct pulsations in his epigastrium. In the region of the pulsations there was felt a firm, smooth, nontender mass in close apposition to the aorta, moving with each pulsation. No expansile pulsation could be made out in this mass, which measures 7 cm. x 5 cm. It did not move with respiration. Gurgling sounds were heard over the stomach area and the region of the mass, which was at about the anatomical location of the pylorus of the stomach.

Examination of urine was negative. On admission his hemoglobin was 25 per cent.; red blood cells, 1,392,000; white blood cells, 5200, with marked variation in size and shape and occasional polychromatophilia.

Wassermann on blood was negative.

Gastric analysis showed a positive guaiac reaction and microscopic blood.

Stool was black, formed, tarry. Guaiac positive.

Roentgen Examination. Unimportant.

Course. On the day of admission the patient ran a slight fever, the temperature ranging from 99° to 100°, but thereafter showed a daily range from 97° or slightly above to 99° or slightly above and the pulse rate for the most part varied between 70 and 80, and the respiratory rate between 20 and 25, until the day before exodus. The stools continued to show occult blood. On July 1 the patient vomited black blood clots, about 4 liters in all. Earlier that morning he had passed stools, consisting of black cast-like clots showing peristaltic impressions. His hemoglobin fell somewhat but his red blood cells remained unchanged. He became blanched and weak, dying within a few hours of respiratory failure.

Autopsy was performed eleven and a quarter hours postmortem. The body was that of a man, aged thirty-nine years, well developed, emaciated. The heart showed a chronic adhesive pericarditis, a moderate degree of fibrous myocarditis. The left coronary artery appeared normal. Just distal to its origin in the right coronary there was found a small, red, irregular, elevated mass about 0.5 cm. in diameter, composed of a friable laminated tissue, on the surface of which fresh fibrin was deposited. Its lumen was dilated at this point. Histological examination showed the intima to be thickened and hyaline, the surface of the intima ulcerated and covered with fresh fibrin.

The lungs, liver, spleen, and pancreas showed no lesions of importance. There was some edema of the kidney. A moderate

degree of arteriosclerosis was found diffusely distributed throughout all these organs. There was a marked secondary anemia.

The lesions in the gastro-intestinal tract were striking. The stomach was collapsed, normal in appearance. On opening it was found to contain a small amount of blood. The mucosa was normal. The duodenum was distended and firm, apparently containing a mass within its lumen. On opening a large blood clot forming a perfect cast of the lumen was found. The ampulla

FIG. 1.—Case I. Anterior wall of aorta, showing situation of aneurysm.

was patent. The mucosa was everywhere normal except for two small ulcers on its posterior wall, the first occurring 5 cm. below the ampulla, the second 2 cm. below the first. These ulcers were 1 cm. in diameter, their edges thin, greenish, friable, indicating an acute necrotic process. There was no induration of their edges. The posterior wall in which these ulcers were situated was bulging into the lumen lying directly upon an indefinite mass. The two ulcers were directly connected with this mass. The greater portion of the jejunum was filled with clot similar to that in the duodenum.

The aorta presented the following conditions: There were no marked lesions up to a point 1.5 cm. below the renal arteries. Here its wall was thinned and wrinkled, there being a marked fusiform dilatation involving the part lying between the renal arteries and the bifurcation. The circumference at the level of the inferior mesenteric artery was 4.5 cm.; at the level of the renal arteries 3 cm. The length of the dilatation was 5.5 cm. In the region of the inferior mesenteric artery there was an ulcerated

FIG. 2.—Case I. Duodenum, showing points of rupture in posterior wall.

oval area 3 x 3.5 cm., the long axis horizontal. The tissue at the base of this ulcer was dark brown, friable, mealy. Extending from the edges there were several bright red granulation-like areas. The origin of the inferior mesenteric artery could not be determined. Anterior to this dilatation there was a soft oval mass which pushed forward the posterior wall of the duodenum. Section antero-posteriorly through this mass showed it to be an aneurysmal sac, its walls being in direct continuity with the aorta. Anteriorly in the region of the duodenal ulcers its wall was thinned. This

sac was completely filled with thrombus, so that it was fairly firm. The opening of the aorta into this sac measured about 1 cm. in diameter. Several parallel sections showed that this perforation and the aneurysmal sac were in no way connected with the inferior mesenteric artery, which was found to lie to the left embedded in the mass of fibrous tissue about the sac. This artery was dilated and tortuous in its beginning and completely occluded by thrombus. Histological examination showed the intima of the aorta

FIG. 3.—Case II. Anterior wall of aorta, showing location of aneurysm.

to be hyaline; media hyaline and degenerated. The walls of the sac were fibrous, containing small follicles of lymphoid cells. The sac was filled with thrombus.

CASE II.—This case we are able to report through the interest of Dr. S. B. Wolbach who turned over the specimen to us and procured copies of the history and autopsy protocol and permission to report the case from the staff of the Long Island Hospital.

J. T. S., male, aged eighty-one years, entered Long Island Hospital, April 25, 1904. Service of Dr. J. J. Minot.

Family History. Negative.

Past History. Had measles and pertussis in childhood. Otherwise has always been well.

Past Illness. Began nine weeks previous to entrance, with a sharp pain in the small of his back; this laid him up so that he was unable to work. When the pain disappeared he usually felt weak His back was not stiff, he had extreme pain on rising or sitting. His appetite was poor. His bowels were constipated. He denied venereal disease.

FIG. 4.—Case II. Duodenum, showing site of rupture.

Physical Examination showed a fairly well-developed, thin, senile man. He had a marked arcus senilis. There was considerable sclerosis of the radial artery. His lungs, heart and extremities were unimportant.

Abdomen. In epigastric region, just above the umbilicus, was felt an oval tumor about 3 x 11 inches in size. The tumor pulsated and was slightly tender. Liver not enlarged. Spleen not palpable.

Urine examination negative.

Course. May 11, sixteen days after entrance, the patient was in fair general condition. His only complaint was of pain in his

back. During the night of May 12 he fell out of bed, after which he vomited and passed by rectum considerable blood. He felt weak, but his pulse remained low. The following morning he was comfortable. During the night of May 16 he collapsed, apparently having been comfortable up to this time. He did not respond to treatment, but grew weaker, and died within half an hour.

Autopsy was performed ten hours postmortem by Dr. George B. Magrath. The body was that of a man, aged eighty-one years; well developed and fairly well nourished.

The heart showed advanced chronic fibrous myocarditis. The coronaries were sclerotic and dilated in the upper part of their course. The lungs showed emphysema, edema and congestion, chronic tuberculosis, and chronic adhesive pleuritis. The liver, spleen, and pancreas showed no lesions of importance. The kidneys on gross examination showed a chronic nephritis. The prostate was hypertrophied. There was marked general anemia.

The lesion of the gastro-intestinal tract were similar to those found in the first case. The stomach was distended with fluid contents which on section was found to consist of semifluid with partially clotted blood. The largest mass of clot was about 15 cm. in diameter. The mucosa was normal. The duodenum and greater part of the ileum were markedly distended with dark red semifluid, and partially clotted blood. The third portion of the duodenum in the midline projects forward, apparently overlying a vague irregular mass about 7 cm. across. The overlying peritoneum was thickened. Upon section at this point the duodenum presented on its posterior wall an opening 2 x 1.5 cm., which on pressure yielded a small amount of grayish-red clot. The edges of this opening were rounded, somewhat irregular, and drawn downward. Upon further dissection this opening communicated with blood clot, and in continuity with the aorta through an opening in its anterior wall 4.5 x 3 cm., the long axis vertical, the edges rounded and infiltrated. This intervening sac, about 5 cm. in diameter, had on its inner surface a laminated clot. Its cavity was for the most part empty. A probe inserted into the opening in the duodenum entered this cavity obliquely. Adjoining this sac and communicating with the aorta was an oval opening 3 cm. in diameter. To the right of and in continuity with the opening in the aorta already described was another nearly empty cavity, 4 cm. in diameter, lined by laminated clot, bounded in front by the inferior vena cava, behind by the lower lumbar vertebræ, and externally by the psoas muscle. This muscle was pale brown, streaked with yellow, and mottled with areas of necrosis and hemorrhage in its lower and posterior portion.

The aorta, in addition to the aneurysms above described, which lie 4 cm. below the renal arteries, presented marked arteriosclerosis

with dilatation. The circumference at the level of the celiac axis was 6.5 cm.; in the middle of the thoracic segment 8 cm.; at the level of the left subclavian artery 7.5 cm. The inner surface was marked by alternating areas of elevation and depression, the latter calcareous, the former soft in some instances, on section containing a mortar-like substance and overlaid with fragile thrombi. Many of these areas were ulcerated. The inferior mesenteric artery was embedded in a mass of firm, fibrous tissue to the left of the aneurysmal sac. On section it was found completely thrombosed, its point of origin to the left of the opening in the aorta.

Discussion. It is interesting that these two cases should be so nearly alike both in their clinical and pathological features. The situations and points of rupture are identical. The same etiological factor is present in each case. Arteriosclerosis as the basic cause in the first case may seem questionable, but the lesions are characteristic, both grossly and microscopically. Furthermore, the clinical findings are against syphilis, the heart condition against an embolus as the primary cause. The etiology in the second case is beyond doubt, owing both to the age of the patient and the character of the aorta.

To account for the history of a month's duration of hemorrhage in the first case there is no definite pathology. Undoubtedly what occurred was that the throbbing pressure of the aneurysm upon the duodenum first wore a small perforation through the intervening walls, which was plugged easily with clot after some hemorrhage. The constant throbbing force, however, made it impossible for complete repair to take place, but rather to hinder it, and at the same time to destroy more of the limiting wall, so that the perforation finally became so large and the flow of blood through it so rapid that no clot could form and check the hemorrhage into the duodenum. In support of this is the fact that the edges of the ulcers were acutely necrotic, containing no evidence of scar formation.

In the second case the severe pain in the back can be explained by the pressure on the necrotic psoas muscle. In this case the walls intervening between the duodenum and sac were thinner than in the first case, so that when the rupture took place a larger opening resulted, with a more acute exodus.

In conclusion we would summarize that these two cases are cases of aneurysm of the abdominal aorta apparently of arteriosclerotic origin which ruptured into the duodenum.

We owe many thanks to Dr. Christian and Dr. Goodpasture for permission to report the case which occurred in this hospital, and to Dr. J. J. Minot for the opportunity and permission to report the second case, which occurred in the Long Island Hospital.

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RELATION OF CONGENITAL SYPHILIS TO MENTAL DEFICIENCY.[1]

By William H. Higgins, M.D.,
RICHMOND, VA.

The general practitioner seldom receives a more unwelcome visitor than the mentally deficient child. The failure of the usual lines of treatment and the hopeless prognosis are too well known for discussion.

It is conservatively estimated that there are 5000 defective children in the State of Virginia. This number, of course, does not include the borderline cases nor the so-called latent types of enfeeblement. Those who have studied the problem most deeply, and who are best able to recognize the moron, usually consider that about 2 per cent. of the school population is feeble-minded, and the larger part of these belong to the high-grade class. It is generally conceded that of these 80 per cent. spring from families in whose lineage similar types may be found.

Naturally, syphilis has been considered as a possible causative factor for many years. In 1888 Shuttleworth examined 1000 cases of mentally deficient children by clinical methods alone and found evidences of syphilis in only 10. Others following him reported similar experiences. Even so recent a writer as Goddard[2] states that if ever syphilis produces feeble-mindedness, it does so only under most favorable conditions. Contemporaneous with this statement, Nonne[3] found syphilis affecting the nervous system in less than 2 per cent. of his series of 1000 cases of nervous diseases.

More recent studies, however, have absolutely established the predisposition of the Treponema pallidum for nerve structures.

[1] Read before the Medical Society of Virginia at its Forty-seventh Annual Session, held at Norfolk, Va.

[2] Feeble-mindedness; its Causes and Consequences, New York, 1914.

[3] Syphilis and the Nervous System, 1913.

Since the valuable contributions of Ravant,[4] Altman,[5] Gennerick[6] and others it is now well known that the spinal fluid is involved early in practically every specific infection. In fact, with the almost universal dissemination of the infectious agent it is difficult to conceive how any organ escapes the infection.

With such a direct relationship existing between acquired syphilis and the central nervous system the frequency of congenital syphilis manifesting itself in its points of predilection should be of vital interest to us. The method of transmission of the spirochete from parent to child has been the subject of discussion for a number of years. Suffice it to say that in the light of our present knowledge the weight of evidence favors maternal transmission. Thus the offspring becomes directly infected with the organisms circulating in its blood. It is important to know that the inheritance of syphilis may take place, although the parental infection has occurred many years before. Nonne[7] quotes a case of Fournier's in which the interval was fifteen years. It may manifest itself in early life in accordance with the usual signs, or the child may escape and later become a victim of the so-called latent or tardy stage.

The pathology in no way differs from that found in acquired infections. The disease may implicate the bloodvessels, meninges, or parenchyma. However, owing to the proximity of these structures and the protean manifestations of the organism it is not likely that one part would be involved without the others showing in the pathological process. The severe forms of restrictive development of the central nervous system are very important and to a certain extent characteristic of congenital lues. According to Nonne, malformations of the brain and cord not infrequently occur, as well as developmental deficiencies in the peripheral nerve roots. Jarisch has demonstrated at autopsy an actual decrease in the number of ganglion cells, a change in the protoplasm of the cells, and vitreous masses around the bloodvessels in the central nervous system. In a comprehensive report of 100 cases of late syphilis, Veeder[8] found lesions of the nervous system in 47, or approximately one-half. Of these 23 were mentally deficient, 14 had cerebrospinal syphilis, and the remaining were divided among less frequent nervous conditions.

There may be a second way by which syphilis may affect the offspring. Quoting from Haberman:[9] "Such parents are themselves constitutionally affected by the disease, and hence the germ cells uniting to form the embryo may also, one or both, be affected, depleted or invalidated, just how cannot be said. The fact remains that this embryo, without containing the infective organism itself, enters from the start on a darkly checkered career whose fate may

[4] Quoted by H. F. Swift: Am. Jour. Med. Sc., 1916, clii, 490.
[5] Ibid. [6] Ibid. [7] Loc. cit.
[8] Am. Jour. Dis. Child., 1914, viii, 283.
[9] Jour. Am. Med. Assn., 1915, lxiv, 1141

be almost as sinister as that of the really infected embryo." Such children will give no Wassermann reaction. They are not syphilitic but are neuropathically or psychopathically marred. Although this modus operandi is open to question it may seem to explain the negative Wassermann reactions sometimes found in the defective children of syphilitic parents.

The first important contribution bearing on the relation of congenital syphilis to mental deficiency was in 1914 by Fraser.[10] In 99 feeble-minded children she found positive Wassermanns in 44.9 per cent. In only 8 of her series were any clinical signs of syphilis present. About the same time Watson[11] in an examination of 204 cases found that 60 per cent. gave a positive reaction. According to some investigators, serological tests of the entire families in which defective children appear show that syphilis is associated with a still higher percentage of cases than is ascertained by the examination of the patient alone.

In Gordon's[12] series of 75, which he studied especially from the stand-point of hereditary syphilis, mental defects of various degrees were present. He found a positive Wassermann in 50 per cent. of his cases. The spinal fluid was examined in 17, all of which reacted positively, with the exception of 3 cases of feeble-mindedness associated with functional nervous disorders.

The object of this paper is to report the results of the serological studies on the first 50 cases admitted to the psychological clinic of the Medical College of Virginia during the past year. The material for this clinic is drawn largely from the retarded classes of the Richmond Public schools, the juvenile court, and other agencies interested in the social welfare of the city. They were sent for the purpose of obtaining the estimate of their mental development as well as a clue to any factors influencing their mental and moral stamina. In addition to the usual physical and neurological examinations, various psychological tests were employed, the results of which are obviously unnecessary in this review. In this series 21, or 42 per cent., gave a positive Wassermann reaction. Their ages varied from seven to sixteen years, and with the exception of 1 all were white. In the accompanying chart of the positive cases the mental level obtained by the Binet-Simon questionnaire, general health, physical defects, and type of temperament are noted. It will be seen that the series is composed largely of the middle and high-grade imbecile and only exceptionally with the low-grade idiot. This in part may account for the relatively low percentage of Wassermanns found in institutions for the feeble-minded. With two exceptions the general health of the children was uniformly good, a fact which made it difficult for us to impress upon the families the necessity for persistent treatment.

[10] Jour. Mental Sc., October, 1913, p. 234.
[11] Ibid.
[12] Gordon: Arch. Pediat., 1916, xxxiii, 273.

Quite at variance with other reports is the striking relative absence of congenital syphilis or organic lesions of the nervous system. Approximately one-half showed a general glandular enlargement. Defects of vision, tonsils, etc., were found no more frequently than in the non-luetic series. The most interesting and suggestive physical aspect was the malformation and caries of the teeth. One of three types were present in each of our cases. The most frequent form was the small, widely separated teeth, with well-marked serrations on the upper and lower incisors. This was associated with moderate caries, especially about the neck of the tooth. The second type was similar to the first, but with a much more advanced grade of dental infection. The least common type was the usual set of teeth riddled with areas of decay. In only 2 of the 21 cases were the teeth even approximately normal. No typical Hutchinson teeth were observed.

Temperamentally the series presented an interesting picture; 14 of the 21 were either incorrigible, disobedient, or displayed fits of temper unlike those usually seen in the normal child. Although our series is too small from which to draw definite conclusions, we are not disposed to feel that the listless, low-grade idiot is so likely to be of syphilitic origin as the high-strung, passionate child with wayward tendencies. It has been shown that the percentage of positive Wassermanns is materially lessened after the sixteenth year.

The recognition of syphilis as a factor in the production of mental enfeeblement is of the uttermost importance in their proper handling and treatment. According to Gordon, where gross malformations do not exist, encouraging therapeutic results are obtained. Nonne[13] speaks of various organic and functional nervous disorders in children with inherited syphilis who improved and some recovered after antisyphilitic treatment. Fournier, likewise, has reported satisfactory results in the treatment of the vague nervous and psychic disorders of congenital syphilitic origin.

If it can be established that inherited syphilis is a frequent factor in the production of the subnormal mentalities the problem of the feeble-minded will present a more hopeful outlook. Recalling the pathology of syphilitic lesions of the brain, it is useless to hope for an amelioration of symptoms in those cases in which the process is long-standing or is destructive in character. Therefore the early recognition of the condition is imperative and our attention should be directed more especially to those borderline types in which mild mental defects are associated with moral delinquencies. It must be borne in mind that the syphilitic organism can be readily transmitted to the third generation and that only a very small percentage of congenital syphilitics can be recognized by clinical methods alone, and no study of an enfeebled child should be considered complete without a Wassermann test. If a negative result is obtained, further

[13] Loc. cit.

inquiry and examination should be made of the parents and other members of the family. Should we be able by this means to reclaim even a few of those destined to become a burden to society and the State our efforts will not have been in vain.

No.	Age.	Mental age.	Years back.	General health.	Physical defects.	Temperament.
1	12	7.6	4.4	Negative	Teeth and ears	Sensitive.
2	16	10.0	6.0	Negative	Teeth	Sexual pervert.
3	11	6.2	5.0	Negative	Internal squint	Incorrigible.
4	15	10.0	5.0	Negative	Teeth	Sullen.
5	12	8.2	4.0	Negative	Glands	Incorrigible.
6	8	4.2	4.0	Negative	Teeth; glands; squint	Incorrigible.
7	7	4.1	3.0	Negative	Teeth	Incorrigible.
8	10	5.2	5.0	Negative	Teeth	Listless.
9	11	7.2	4.0	Negative	Teeth; glands	Disobedient.
10	12	10.0	2.0	Negative	Negative	Incorrigible.
11	14	10.0	4.0	Negative	Negative	Incorrigible.
12	15	10.0	5.0	Negative	Glands	Slow.
13	12	10.0	2.0	Headache	Glands	Temper.
14	14	7.3	6.7	Negative	Heart glands	Incorrigible.
15	11	4.0	7.0	Headache	Teeth; ears	Listless; incorrigible.
16	14	10.4	3.6	Negative	Glands; teeth	Incorrigible.
17	8	6.3	1.7	Negative	Glands; teeth	Slow.
18	11	7.2	3.8	Negative	Teeth	Slow.
19	15	10.8	4.2	Negative	Teeth; speech	Sullen.
20	10	5.0	5.0	Negative	Teeth; glands	Sex; habits; incorrigible.
21	16	8.0	8.0	Negative	External squint and teeth	Slow and sullen.

MULTIPLE SEROSITIS; REPORT OF A CASE WITH AUTOPSY FINDINGS; DISCUSSION OF ITS CLASSIFICATION.

By George H. Evans, M.D.,

ASSISTANT PROFESSOR OF CLINICAL MEDICINE, UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL, SAN FRANCISCO, CALIFORNIA.

Since the publication of the interesting and exhaustive monograph of Kelly on this subject in 1903, very little has appeared in medical literature in reference to the condition known as chronic obliterative pericarditis with ascites.

Chronic perihepatitis, associated with varying degrees of chronic inflammation of the pericardium and pleuræ, as a local manifestation of diffuse chronic peritonitis has been recognized for more than half a century. Rolleston restricts the name multiple serositis to this condition, and includes in this category the chronic hyperplastic perihepatitis described by Curschmann in 1884 and called by him Zuckergussleber, sugar-iced liver, so-called from the thick encasement of fibrous connective tissue, more or less enveloping the liver, whitish, glistening, somewhat resembling confectioners' icing.

In 1896 Pick described, under the term "pericarditic pseudo-liver cirrhosis," three patients whose ailment was predominantly ascites of long duration together with enlarged liver, but at autopsy in each instance showed thickened and obliterated pericardium not suspected during life. Two of these cases presented some degree of liver cirrhosis, not in proportion, however, to the amount of ascites present, the other an enlarged nutmeg liver, the cardiac liver of the French writers. All of these cases presented marked perihepatitis. This communication became the subject of much discussion at the time, and the condition described has since been frequently called Pick's disease, though, as mentioned before, it was known to earlier writers.

Rolleston distinguishes the condition described by Pick from the cases of perihepatitis which he considers as only a local manifestation of diffuse chronic peritonitis, in that the former presents primarily an extreme grade of chronic venous engorgement of the liver dependent upon the cardiac condition, the peritoneal thickening or adhesions that may occur being incidental. Thus we have portrayed two more or less distinct clinical pictures: the so-called Pick's disease, in which the predominating condition is the nutmeg or cardiac liver, characterized by chronic venous engorgement, later by red atrophy, and ultimately cirrhosis; on the other hand, those cases of multiple serositis in which the perihepatitis is the prominent clinical feature. Both conditions present at autopsy pericardial lesions, though this condition may not have been apparent during life.

Much confusion has naturally arisen through this rather delicate distinction.

Kelly, in 1903, attempted a broader classification, and grouped under the name multiple serositis those cases "characterized clinically by marked ascites, with little or no edema of the legs, cases in which the diagnosis of cirrhosis of the liver is usually made and which at the necropsy reveal chronic obliterative pericarditis and certain morbid changes in other tissues and organs, notably pleuritis, peritonitis, perihepatitis, nutmeg liver, red atrophy of the liver, cirrhosis of the liver, etc."

In the monograph the author has gathered from the literature 39 cases which he has tabulated with reference to symptomatology and autopsy findings. A complete bibliography on the subject up to 1903 is appended, to which the reader is referred.

It is of interest in the present discussion to report the following case, the clinical notes for which I am indebted to my intern, Dr. M. J. Price:

C. J. D., single, male, aged fifty-four years, a native of Panama, was admitted to my service at St. Luke's Hospital, June 22, 1916, complaining of cough, increasing dyspnea on exertion, and swelling of the extremities, which had lasted for the last nine months.

There was nothing of importance in his family history.

The patient's general health had never been very good, and as a child he was subject to frequent colds. Had never been subject to sore throat. At the age of eighteen he had an acute attack of inflammatory rheumatism, involving all the joints, and persisting for a period of three months. There had been no recurrence of these symptoms. Fifteen years ago he was confined to his bed for six weeks with typhoid fever, making an uneventful recovery.

Respiratory System. Has been subject to frequent colds, but denies pain in the chest, hemoptysis, and night-sweats.

Cardiovascular System. Nothing abnormal up to nine months ago.

Urinary System. Denies dysuria, polyuria, and nycturia.

Sexual System. Single; denies Neisser and luetic infections.

Neuromuscular Systems. Nothing abnormal.

Weight. No recent change in weight; 114½ pounds.

About September, 1915, patient noticed that he was gradually becoming short of breath on the slightest exertion. This symptom gradually increased and was accompanied by swelling of the extremities. Within the next month the patient developed a severe, fatiguing cough, which has lasted up to the present time.

During the month of January, 1916, patient was very ill and weak. The distressing symptoms at that time were shortness of breath, swelling of the ankles, and a severe dry cough. Last week he was seized suddenly with a temporary paralysis involving both extremities and was unable to use them for the greater part of a day.

Man of medium build, five feet five inches tall, dark complexion, and of fair development and nourishment.

Condition of the mouth unhealthy; many diseased teeth and considerable pyorrhea. No noticeable abnormalities of nose, ears, and throat.

The cervical glands are palpable and small. No palpable enlargement of the axillary and clavicular glands.

Heart. The apical impulse is visible 13.5 cm. from the median line. The heart sounds are indistinct and muffled; the pulmonic second sound is accentuated. There are no murmurs. Peripheral arteries do not appear thickened.

Blood-pressure. Systolic 120, diastolic 90, pulse rate 90.

Lungs. The excursion of the diaphragm is diminished on both sides. There is hyperresonance over both lungs to the fourth ribs, from there down dulness or flatness on both sides, shifting somewhat on change of position of the patient. Auscultation reveals a few crepitant and sonorous rales above the areas of dulness. Vocal resonance and tactile fremitus are absent over these areas and the breath sounds are faintly heard. Posteriorly dulness corresponds with that in front, with similar signs on auscultation. Respirations 24. Roentgen-ray examination confirms the physical findings, but

shows in addition a heavy right hilus shadow and a dilated descending aorta.

Abdomen. The abdomen is symmetrical and there are no areas of tenderness or rigidity. The hepatic dulness extends from the fifth rib and is just palpable at the costal margin. The edge is smooth. The spleen is not palpable and by percussion seems of normal size. There is a small amount of ascitic fluid.

Extremities. Marked pitting edema of both feet and legs. The finger-joints show evidence of hypertrophic arthritis.

Genitals. No apparent abnormalities.

Skin. A peculiar brownish maculopapular eruption is present in the clavicular fossæ, and on the right thorax a similar eruption follows irregularly the course of the seventh intercostal nerve.

Urine. Scanty, sometimes turbid, traces of albumin, indican in excess, with hyaline and occasional granular casts. Kidney function as shown by the phenolsulphonephthalein test practically normal. Blood findings negative, except for a slight increase in the leukocytes (12,430) with a relatively high lymphocyte count. Blood serum presented a negative Wassermann. The sputum, scanty in amount, reveals no tubercle bacilli on repeated examinations, though it contains various other bacteria and several herzfehlerzellern. Cutaneous and intracutaneous tuberculin tests were negative.

He remained in the hospital until the time of his death, about one month later. The edema in the legs promptly went down following rest in bed, and the dyspnea and cough improved. Thoracentesis was done three times and considerable amounts of fluid aspirated, once from the right and twice from the left pleura. Removal did not affect the position of the apex beat, nor was it changed on change of posture of the patient. Following the removal of 650 c.c. of fluid from the right pleura obliteration of the cardiohepatic angle was demonstrated. The fluid removed was slightly turbid, not bloody, and was negative to cultures and guinea-pig inoculations.

The urine was persistently low in quantity, though at first much increased by the administration of theocin.

He became progressively weaker, was very nervous and restless the last few days of life, and died in an attempt to get out of bed.

Clinical Diagnosis. Chronic pleurisy; chronic pericarditis, with effusion; pericardiopleural adhesions; probable chronic peritonitis.

Autopsy. July 29, 1916. Made by Dr. E. V. Knapp, the hospital pathologist, showed the following:

Man about forty-five years old; dark complexion.

Pupils dilated and equal; no jaundice; visible mucous membrane pale.

Teeth poorly kept.

Marked emaciation; no edema.

Dark brown pigmented areas over lower abdomen and lower thigh and over side of chest, especially the right; no infiltration or induration. Over the lower thighs, patellæ, shins, forearms, especially the back, there are numerous pustules, about 2 mm. in diameter; dark brown induration around them; center is depressed, slightly indurated, somewhat shotty. None on soles of feet or palms, none on chest or face. Pigmented areas scattered over buttocks and lower lumbar region. No glandular enlargement except inframandibular, which are hard and shotty.

Median incision shows a moderate amount of fat of good color; the blood is bright red. Cartilages and muscles are of good color. There is a large amount of slightly turbid, blood-tinged fluid in the abdominal cavity.

The diaphragm is at the fifth intercostal space on each side.

The liver is about four finger-breadths below the costal margin in the midclavicular line on the right side. The left lobe is about three finger-breadths below the costal margin in the midclavicular line. The notch is well marked.

The intestines and omentum are slightly congested.

There are no abnormalities of the sternum.

Both pleuræ contain a large amount of hemorrhagic fluid.

The lungs are somewhat atrophic; there are no adhesions at the right apex; there are many adhesions at the left apex and hilus, also posteriorly. There are many fibrinous adhesions at the right upper and lower lobes attached to the pericardium.

The pericardial sac is filled with a considerable amount of straw-colored fluid. There are numerous pleuropericardial, mediastinopericardial and phrenopericardial adhesions. The thoracic wall is smooth.

The right lung shows many interlobular fibrinous adhesions. The surface of the right lung is moderately smooth; there is a moderate amount of dark brownish pigmentation. There are no adhesions or scars at the apex. In the upper lobe, anteriorly, there is a hard, indurated smooth mass about the size of a small hen's egg. Middle lobe is free, with the exception of a small nodule. At inferior lobe there is a hard, nodular, indurated mass, about the size of a lemon, somewhat hemorrhagic. On cut section this mass is glistening and dark red in color and definitely outlined. Other nodules are similar. The pulmonary vessels are clear.

The left lung shows many fibrinous adhesions at the apex and upper margin of the lower lobe; surfaces are smooth; the left lower lobe is congested and indurated and of a dark muddy color. The cut surface appears deep red; is only slightly crepitant.

Amount of fat about the heart is moderate; the muscle tissue is flabby, somewhat pale, and brownish; surfaces are smooth. The aortic valve is slightly roughened but shows no vegetation or calcification. Other valves are normal. The left ventricular wall is about 3 cm. thick, the right about ½ cm. thick. Trabeculæ are

hypertrophied. There is no sclerosis in the coronary arteries. The pericardium is smooth, thick, and glistening; there are no adhesions to the myocardium.

The heart is about 18 cm. across at the base, 16 cm. from base to apex, and about 10 cm. through.

There are many adhesions between the left lobe of the liver and the lesser curvature of the stomach, between the right lobe and the diaphragm and between the right lobe and transverse colon. The gall-bladder is normal in size. The liver is about 24 x 15 x 10 cm. The surface is smooth; the cut surface has a brownish, mottled appearance; the lobules stand out very distinctly. There are no areas of induration or nodules. The liver capsule is somewhat thickened.

The lower pole of the spleen is at the eighth rib in the axillary line. There are a few adhesions between the spleen and the greater curvature of the stomach. The spleen is about 10 x 7 x 4 cm., of a grayish-slate color. Cut section shows the trabeculæ well marked, the pulp pale.

The right suprarenal is pale yellow, flabby, but there is no evidence of tuberculosis. The left is normal in size and the surface is slightly nodular.

The kidneys are normal in size and the capsule stripped easily. The right shows the cortex somewhat congested and a small scar at the lower portion, anteriorly, about the size of a pea. The left presents six small cysts, each about the size of a pea, which contain a clear yellow fluid. At the lower middle portion there is a depressed scar about the size of a dime. There is some atrophy of the cortex; the glomeruli and tubules are prominent.

There are many adhesions between the pancreas and the upper part of the stomach and duodenum. It is normal in size and appearance.

The stomach is normal in shape and position. There are a great many adhesions to the surrounding organs; the omentum is normal.

The appendix is normal and retroverted.

There are many adhesions binding the transverse colon and the upper intestines.

The lower large bowel is firmly adherent.

There are no areas of induration in the lower rectum.

The prostate is small and fibrous.

The anterior and posterior surfaces of the bladder are firmly bound to the surrounding tissues by dense bands of fibrinous tissue.

There are a few scattered yellowish plaques in the upper portion of the aortic arch.

Histological Examination. Sections of the spleen show normal markings. There is a slight interstitial thickening of the pulp.

Sections of the liver show a slight thickening of the periportal connective tissue. There is slight cell infiltration around some of

the bile capillaries. The capsule also shows slight connective-tissue thickening. There is a considerable amount of fat in the periphery of some of the lobules in the form of small and large droplets in the liver cells. Near the centers of the lobules the liver cells are small and contain much fine, brownish pigment.

Sections of the lungs show in places marked hyperemia. Many of the air spaces are filled with erythrocytes and desquamated epithelium. Another section of the lung shows an area in which the air spaces are filled with blood. Around this area is a marked hyperemic zone.

Sections of the pancreas are normal.

Sections of the heart muscle show areas of intense cell infiltration, with leukocytes and partly organized blood clot.

Sections of the suprarenals are normal.

Sections of the kidneys show numerous scars in the cortex. There is a moderate amount of fibrinous thickening in the straight tubules. Many waxy and granular casts in the tubules. There is a small pyramidal area in the cortex in which the glomeruli and some of the tubules are apparently necrotic.

Sections of the skin in the region of a pustule show no evidence of inflammation, but the epithelium and subcutaneous tissues are entirely necrotic.

Anatomical Diagnosis. Polyserositis. Pleurisy with adhesions. Pericardiodiaphragmatic (pleural) and mediastinal adhesions. Pericardial effusion. Chronic peritonitis. Adhesions of bladder, intestines, pancreas. Myocardial hypertrophy and degeneration. Chronic congestion of liver. Beginning cirrhosis. Arteriosclerosis involving aorta and kidneys. Pulmonary infarction.

While the case here recorded presents the widespread disease of the serous membranes which justifies the diagnosis of multiple serositis, a critical analysis of the clinical course and the autopsy record makes it at once apparent that it presents features somewhat at variance with the descriptions of other investigators.

Ascites was not a prominent condition. While it was demonstrable at examination and a considerable amount of fluid was found at autopsy, it had not been in such amounts as to require tapping for its relief, and could scarcely be said to fall within the classification requirements of Kelly, who describes marked ascites as the striking clinical feature in all of the 39 cases reported by him. That the degree of ascites will vary dependent upon the pathological condition of the liver is certain. Chronic venous engorgement of the liver is present in a majority of these cases, though cirrhosis is found in some. It is questionable upon what the ascites depends. The sequence of appearance of the pathological features is uncertain. It is open to question whether the ascites is produced by the chronic portal obstruction dependent upon the venous engorgement of the liver or whether a chronic peritonitis caused by portal and peritoneal engorgement is responsible for it.

Again, the perihepatitis, marked in a majority of the cases reported, particularly if it leads to compression of the liver and the portal radicles, produces an important factor contributing to ascites. The clinical history of these patients throws little light on the organs or tissues first affected in multiple serositis. Pick considered the obliterative pericarditis the primary condition and sought to establish an etiological relationship between it, the enlarged liver, and the ascites.

Chronic Obliterative Pericarditis. This condition next to ascites is the most constant. It was present in all of the cases presented by Kelly—complete obliteration in the majority of them. It was present in all of Pick's cases. Other writers, however, have not found it constant. Rolleston, who makes a sharp distinction between pericarditic pseudocirrhosis of the liver (Pick's disease) and multiple serositis, admits its frequency in the latter disease, but does not assume its importance as the primary pathological factor. Picchini found the pericardium involved in only 9 of 50 cases observed by himself.

There were no adhesions between the pericardium and cardia in the case presented by the writer. There was pericardial effusion. The ravages of inflammatory disease of the pericardium were, however, marked by numerous adhesions of the pericardium to the mediastinum, pleura, and diaphragm. The etiology responsible for these cases of pericarditis obliterans is rather obscure and the antecedent pathology is frequently uncertain. As long ago as 1837 Laennec called attention to the fact that the adhesions followed the absorption of pericardial effusion. In all probability obliterative pericarditis has been given too great significance as the factor in the production of the disease, and the role played by the weakened cardiac muscle has been overlooked. Adherent pericardium may exist symptomless for years. Myocardial weakness, however, dependent upon or coincidental with it, is responsible for the venous congestion of the liver with its resulting chronic portal obstruction, ascites, and peritonitis.

Chronic perihepatitis is constant, varying from a slight connective-tissue thickening of the capsule, as described in the writer's case, to the Zuckergussleber of Curschmann, where the liver is encased in a coating of fibrous tissue of cartilaginous consistence. The importance of perihepatitis as the primary pathological factor on which the widespread involvement of the serous membranes depends has been advanced by some investigators.

Of interest here is the association of chronic perihepatitis with arteriosclerosis and granular kidney. Hale White, quoted by Rolleston, found it present in 19 out of 22 cases. There appears to be some connection between arteriosclerosis and fibrosis of the serous membranes. Rolleston admits that arteriosclerotic change in the kidney may be a disposing cause of chronic inflammation in the body generally by reducing the bactericidal power of the blood.

In the case here recorded sclerotic plaques were found in the aorta, and the kidneys showed numerous arteriosclerotic scars in the cortex.

Of interest in this case were the multiple infarcts found in the right lung, two of them of considerable size. This condition has not been heretofore observed in the literature on the subject. It may have accounted for the extreme restlessness and dyspnea which were present before death. Unfortunately no cultures were made.

The cause of multiple serositis is obscure. The tendency to attribute to the tubercle bacillus the role of the etiological factor in chronic pericarditis and chronic disease of serous membranes generally is altogether too prevalent. That tuberculosis is often the cause and that tuberculous lesions are frequently found associated with this condition is undoubtedly true. That evidence of a tuberculous infection can be seen at most autopsies is equally true. No evidence of tuberculosis was found in any of the organs of the case here recorded, nor was there anything in his history suggestive of antecedent tuberculous infection. Of significance, however, was the history of acute rheumatism which he had when eighteen years of age, and which lasted for a period of three months, though he had no subsequent attacks. The case reported by Kelly presented a similar history, and he emphasizes the etiological importance of rheumatism.

There would seem to be considerable confusion among writers concerning the classification of this interesting condition, of which multiple serositis seems to be the one constant feature. This would appear to be largely because certain pathological conditions have been somewhat arbitrarily presented as fundamental to the production of the group of lesions making up the clinical entity. That the microbic invasion responsible may attack any of the structures involved must be conceded. It would therefore appear that until further investigation presents more light on the etiological factor or factors causative that a broader classification would be desirable and that multiple serositis should not be restricted to a clinical picture necessarily associated with adhesive pericarditis or perihepatitis, which, after all, are probably but conditions incident to the general serous involvement rather than responsible for it.

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THE ESTIMATION OF UROBILIN AND UROBILINOGEN IN THE DUODENAL CONTENTS.

By Herbert Z. Giffin, M.D., Arthur H. Sanford, M.D.,
and
Thaddeus L. Szlapka, M.D.,
Mayo Clinic, Rochester, Minnesota.

In a study of diseases associated with evidence of increased blood destruction it is desirable to attempt to estimate the activity of hemolysis and to compare this with the probable capacity for blood regeneration. Many workers have used as an index of blood destruction the changes that occur in the contents of the bile, namely, the amount of bile pigment excreted, and especially the amount of urobilinogen and urobilin. Eppinger estimated the amount of urobilinogen and urobilin in the stools by means of the Charnas spectrophotometric method and stimulated interest in this manner of estimating blood destruction, particularly in pernicious anemia and hemolytic icterus. In this country a more simple procedure for making rough quantitative estimates of these substances has been devised by Wilbur and Addis. Robertson, making use of the method, has reported findings in every way comparable to those of Eppinger.

The theories advanced with regard to the fate of hemoglobin after hemolysis are not well substantiated. Recent investigations by Whipple and Hooper are especially important. Their experiments involved a great deal of careful study over a considerable period of time, and, of necessity, only on dogs. Their conclusions are in some respects iconoclastic. If further work substantiates their findings it may be necessary to change our conception of the factors concerned in the formation of bile pigment. These authors conclude, especially from their results regarding the influence of diet on bile-pigment production, that the disintegration of red blood cells is not the important factor in the production of bile pigments. They have made estimations chiefly of the amount of bilirubin. It will be interesting to learn of their results with respect to urobilin and urobilinogen, for in the clinical studies on the hemolytic anemias there is now considerable evidence from many observers that the amounts of these substances excreted are definitely increased. This increase, it is true, may be the result of impaired organ function as well as of blood destruction. The quantity of bilirubin does not, however, run parallel to the quantity of urobilin and urobilinogen in pathological conditions.

In our own studies we have made use of the modifications of the Wilbur and Addis methods recently devised and reported by

Schneider. The essential advance in technic is the application of the observations, not to stool extracts, but to the contents of the duodenum as collected by means of an Einhorn tube. In this liquid an amount of the biliary pigments and their derivatives sufficient for quantitative determinations is readily obtained in a short time. While an estimation of the amounts of these pigments in the duodenal contents at a given time cannot be regarded as an index of the total amount excreted in twenty-four hours, objections are also advanced with respect to the values obtained in the twenty-four-hour stool in which some proportion of the substances may be destroyed or changed in character. The values obtained by Schneider's method have been so definitely in accord with the clinical manifestations that there is little doubt of the existence of a relationship which it is to be hoped may be made clearer by further study. These values are also in accord with the results obtained from estimations on the stool. The technic we have used, which in all of its essentials is that described by Schneider, is presented in detail, in order that it may be readily understood and easily followed. We wish to express our indebtedness to the originator of this method for his personal interest in our work.

The Method of Obtaining the Duodenal Contents.*

The tube and the metal capsule employed are similar to those of the Einhorn duodenal tube, but experience has led to the use of a somewhat stiffer tube and a capsule which, though similar in shape, is slightly larger and heavier, weighing 6.4 gm. This model of capsule was recommended by Schneider. However, an ordinary Einhorn or a Rehfuss bucket may be used. For convenience in observing the contents of the tube a piece of glass tubing is inserted at its end.

In the preparation for the examination the patient is instructed to partake of no food for at least twelve hours, except perhaps a little tea or coffee without cream, and to take frequent sips of warm water up to the time of the examination. The nature and purpose of the test are also explained, as the patient's confidence and coöperation aid in the passage of the tube.

The passage of the duodenal tube is a simple procedure. The metal capsule is placed on the back of the tongue and the patient is directed to swallow hard several times in rapid succession. There is usually some difficulty as the capsule reaches the level of the cricoid, but this is overcome by deep breathing. After the capsule has passed this irritable zone, peristalsis carries it along without further discomfort to the patient and largely beyond his control. He merely swallows from time to time, taking a few sips of warm water and the capsule finally reaches the stomach and comes to rest at about 60 cm. from the incisor teeth.

* Described by Szlapka.

The patient is now made to lie on his right side, with the hips elevated eight or ten inches. The pyloric end of the stomach thus becomes more dependent, and gravity, aided by gastric peristalsis, causes the capsule to pass through into position in the duodenum—some 70 cm. from the incisors. In our experience this is accomplished in from fifteen minutes to an hour, usually in about forty-five minutes. At no time is it necessary to push the tube on its way. Pushing only tends to coil it up in the stomach and may even frustrate its passage into the duodenum.

With the patient on his side the end of the tube is allowed to hang well over the edge of the table. Gastric contents siphon out first, the siphon being started by the injection of a few cubic centimeters of warm water. As the capsule moves through the pylorus into the duodenum the fluid recovered becomes yellowish and finally a clear bile is obtained. It may vary in color from light yellow to chocolate brown. Pure duodenal fluid is faintly alkaline, clear, of quite uniform color and viscid; the foam is golden. The liquid must be alkaline. Its mere appearance is not a safe guide as to its identity, although with experience one may come to recognize it readily. The character of the fluid collected must be observed closely, as from time to time the pylorus permits the passage of gastric contents into the duodenum. This impure liquid may be detected by its change in color and its dull, cloudy, opalescent appearance, diminished viscidity, more rapid flow, and change in reaction to litmus or Congo red. It should, of course, be discarded.

Should the flow of bile become interrupted an unusual length of time, the injection of a little warm water into the tube or the taking of deep breaths by the patient, will help to reëstablish it. The application of suction is neither necessary nor advisable.

It is our custom to collect the duodenal contents in a small, amber-colored bottle, as air and light cause the rapid transformation of the urobilinogen into urobilin; 20 c.c. of liquid are necessary for the test, which should be made immediately.

Laboratory Technic.*

The duodenal contents is poured into a graduated cylinder as soon as it is brought to the laboratory, and its gross appearance noted. Normally it is a light, straw-colored, viscid fluid, and this is reported as normal yellow (N). The color may vary to dark yellow, brown, and chocolate. The dark-colored fluids always yield much bilirubin, but the color of the duodenal contents does not always indicate the amount of urobilinogen or urobilin present, as these derivatives of bilirubin are sometimes demonstrated in considerable amounts in normal yellow fluids. Occasionally, however, in cases other than pernicious anemia or hemolytic icterus a colorless, watery secretion with no biliary pigments or derivatives is collected.

* Described by Sanford.

When 20 c.c. or more of clear duodenal contents is collected it is divided into two 10 c.c. portions in 25 c.c. graduates. To one 10 c.c. portion is added an equal amount, 10 c.c., of a saturated alcoholic solution of zinc acetate (Schlesinger's solution).* The mouth of the graduate is closed by the thumb and the contents thoroughly mixed by vigorous shaking for about one minute. The mixture is then filtered through a single layer of coarse filter paper, the filtrate being collected in another clean, dry graduate. When exactly 10 c.c. of filtrate is obtained it is used for testing for urobilin and urobilinogen. To this mixture, which consists of 5 c.c. of duodenal contents and 5 c.c. of Schlesinger's solution, is added exactly 1 c.c. of Ehrlich's aldehyde reagent† measured with a 1 c.c. pipette. The color of the fluid is usually significant when viewed by transmitted and reflected light. If urobilinogen is present in considerable amount, especially if it predominates, the fluid, on the addition of Ehrlich's reagent, becomes a cherry red, varying in intensity with the amount of chromogen present. When there is a preponderance of urobilin the color by transmitted light is yellow or brown, and by reflected light a green fluorescence characteristic of mixtures of urobilin with zinc salts is noted. The graduate is now set in the dark for fifteen minutes before it is examined spectroscopically. This length of time seems necessary to sufficiently sharpen the absorption bands of the spectrum, while if the mixture stands for longer intervals of time some of the mother substance, urobilinogen, may become converted into urobilin.

While waiting before making the spectroscopic examination the second 10 c.c. portion is tested for bilirubin. To the duodenal contents in the second 25 c.c. graduate is added exactly 10 c.c. of 10 per cent. aqueous solution of calcium chloride made slightly alkaline to litmus with normal sodium hydrate solution. The mixture is thoroughly shaken and then poured into two 15 c.c. centrifuge tubes, 10 c.c. in each. It is then rapidly centrifugalized for a few minutes to collect the precipitate into a compact mass. The supernatant fluid is decanted and the residue washed out of the tubes with a few cubic centimeters of acid alcohol‡ into a porcelain evaporating dish. In all, about 20 c.c. of acid alcohol is used to dissolve the precipitate. The alcohol mixture is carefully heated on a copper warming stage and allowed to boil vigorously. The color, which may be brick red, soon changes to green if there is much bilirubin present. The mixture is concentrated so that its volume just reaches the "U" in an Esbach albuminometer. Alcohol is added to the mark "R." The color of the fluid by transmitted light is then compared with three standard tubes marked +, ++, +++,

* Ethylalcohol, 500 c.c.; zinc acetate, quantity sufficient for saturation.

† Paradimethylaminobenzaldehyde, 4 gm.; hydrochloric acid, 30 c.c.; distilled water, 30 c.c.

‡ Hydrochloric acid, 5 c.c.; alcohol, 20 c.c.

according to the shade of emerald green as viewed by transmitted light. The standard tubes are prepared arbitrarily from specimens containing appreciable amounts, moderately large amounts, and excessive amounts of bilirubin. These alcoholic solutions may be kept indefinitely without change of color, though it may be advisable to place the tube in the dark when it is not in use.

The first mixture is now examined spectroscopically for urobilinogen and urobilin. The spectroscope we use is of the simple students' type having a collimator, with a slit adjustable by a thumb screw, a scale tube, and a draw-tube type of telescope. The light we use is a 250-Watt Tungsten electric lamp mounted on a stand with a green shade reflector. This is adjusted so that when the collimator of the spectroscope is placed about eight inches from the globe a brilliant spectrum is produced. The glare of the light is kept from the eyes by the shade of the lamp, and by a shield of black cardboard perforated so that it may be slipped on the collimator tube. For observing the absorption bands, Schneider uses a 50 c.c. graduated cylinder. Our own observations were made in this manner up to October 1, 1916; since that time we have used a spectrum cell with parallel sides, and of such dimensions that the distance traversed by the rays of light in passing through the fluid is exactly 1 cm. Schneider opens the slit of the collimator eight half-turns or four full turns of the adjusting screw when using a cylinder for examining the solution. We have found this slit too wide with the standard spectrum cell, and have accordingly used a slit of just half the width. Thus to adjust the collimator we completely close the slit and then open it by four half-turns or two complete turns of the adjusting screw. This gives apparently about the same degree of absorption with the standard cell as is obtained with the cylinder when the slit is twice as wide, so that in this way all readings are made to conform to Schneider's standard.

The presence of urobilin is marked by a broad band in the blue end of the spectrum. The violet rays are completely absorbed, and if there is much urobilin present the entire blue portion and nearly all of the green may be obliterated. Urobilinogen absorbs a narrow portion of the spectrum in the yellow at the edge of the green, and if present in large amounts the band may be broad enough to obliterate the entire yellow portion of the spectrum. It is located by its proximity to the "D" Frauenhofer line while urobilin extends from between the "B" and "F" lines to the violet end of the visible spectrum.

The method used by Schneider to estimate the quantity of the absorbing substances is that suggested by Wilbur and Addis. The solution is diluted carefully with alcohol until the absorption bands disappear. The urobilinogen and urobilin differ in their intensity; consequently the disappearance of the absorption bands will occur with different dilutions, although at times the same dilution causes

the clearing of the spectrum in both regions. The end-point is determined when the absorption band disappears, but can be made out faintly when the slit is narrowed to just half of its former opening; that is, when the cylinder method is used the adjusting screw is turned four half-turns. With the standard spectrum cell the end-point is determined by causing the reappearance of absorption bands with two half-turns.

The amount of urobilin and urobilinogen is estimated according to the Wilbur and Addis method for 1000 c.c. by multiplying the number of dilutions by 200. This factor is used since 5 c.c. of duodenal contents is represented in 10 c.c. of filtrate obtained from the mixture with the Schlesinger solution. The number of units of urobilinogen and urobilin are added together and the total number of units reported, *e. g.*, urobilinogen (three dilutions) $3 \times 200 = 600$ units; urobilin (four dilutions) $4 \times 200 = 800$ units; total 1400 units.

Clinical Observations.*

A total of 119 tests have been made in 89 cases. The results will be considered in three groups:

1. Results obtained in a series of miscellaneous cases of which there were 22, and 22 tests.
2. The findings in hemolytic jaundice, 6 cases, 12 tests.
3. The findings in pernicious anemia, 61 cases, 85 tests.

The study includes the tests made in all cases up to November 15, 1916, with the exception of 9 in which the diagnoses were so obscure as to render the results positively and negatively valueless. Brief protocols of the 22 cases in the miscellaneous group are presented.

1. Miscellaneous Cases.

Anemia from Hemorrhage. Case 1 (157200).—Woman, aged fifty years. Uterine myomas. Hysterectomy. Spleen normal in size but hard; liver congested. Hemoglobin, 30 per cent.; red blood cells, 3,470,000.

Duodenal contents: Color, yellow. Urobilin, 200 units; urobilinogen, trace; total, 200+ units.

Case 2 (164791).—Woman, aged forty-one years. Hysterectomy. Appendectomy. Spleen twice normal size; liver slightly enlarged. Hemoglobin, 35 per cent.; red blood cells, 3,190,000.

Duodenal contents: Color, yellow. Urobilin, 200; urobilinogen, 0; total, 200.

Case 3 (156514).—Woman, aged forty-seven years. Cervical polyp. Melancholia of climacteric. Liver normal; spleen normal, Hemoglobin, 50 per cent.; red blood cells, 3,600,000.

Duodenal contents: Urobilin, 200; urobilinogen, 0; total, 200.

* Made by Giffin.

CASE 4 (160583).—Man, aged fifty-nine years. Bleeding hemorrhoids. Liver just palpable; spleen normal. Hemoglobin, 30 per cent.; red blood cells, 3,340,000.

Duodenal contents: Color, yellow. Urobilin, trace; urobilinogen, trace; total less than 200.

CASE 5 (172166).—Woman, aged twenty-five years. Slight menorrhagia. Liver normal; spleen normal. Hemoglobin, 60 per cent.; red blood cells, 4,460,000.

Duodenal contents: Color, yellow. Urobilin, 500; urobilinogen, 0; total, 500.

Chronic Arthritis. CASE 6 (85456).—Man, aged fifty-one years. Mild arthritis and neuralgia. Liver normal; spleen just palpable. Hemoglobin, 72 per cent.; red blood cells, 4,620,000.

Duodenal contents: Color, yellow. Bilirubin trace. Urobilin, 800; urobilinogen, 0; total, 800.

CASE 7 (131120).—Man, aged fifty years. Mild chronic arthritis; dental abscesses; chronic tonsillitis. Liver normal; spleen normal. Possibility of pernicious anemia. Hemoglobin, 38 per cent.; red blood cells, 1,700,000; white blood cells, 12,200; color index, 1.1. (One year ago hemoglobin 57 per cent.; red blood cells 4,080,000.)

Duodenal contents: Urobilin, 600; urobilinogen, 600; total, 1200.

Dental Abscesses; Anemia of Secondary Type. CASE 8 (162929).—Woman, aged thirty-two years. Liver normal; spleen just palpable. Hemoglobin, 52 per cent.; red blood cells, 4,520,000.

Duodenal contents: Color, yellow. Bilirubin trace. Urobilin, 600; urobilinogen, 0; total, 600.

Cholelithiasis. CASE 9 (151433).—Woman, aged fifty-seven years. Gall-stones. Slight possibility of pernicious anemia. Cholecystectomy. Liver normal; spleen normal. Hemoglobin, 49 per cent.; red blood cells, 2,360,000; color index, 1.

Duodenal contents: Color, yellow. Urobilin, 600; urobilinogen, 0; total, 600.

Syphilis. CASE 10 (153153).—Woman, aged thirty-two years. Syphilis of the liver and spleen. Splenectomy, April 29, 1916; spleen 760 grams. Liver very large with gummas and contractures. Hemoglobin, 49 per cent.; red blood cells, 3,430,000.

Duodenal contents: Color, yellow. Urobilin, 1000; urobilinogen, trace; total, 1000+.

CASE 11 (151226).—Woman, aged thirty-four years. Probable luetic anemia. Some evidence of nephritis. Absence of history of pernicious anemia. Liver normal; spleen normal. Hemoglobin, 45 per cent.; red blood cells, 3,330,000; color index, 0.6.

Duodenal contents: Color, yellow. Bilirubin, trace. Urobilin, 800; urobilinogen, 0; total, 800.

Carcinoma. CASE 12 (169462).—Man, aged forty-five years. Carcinoma of stomach. Roentgen findings of extensive carcinoma. Wassermann negative. Hemoglobin, 29 per cent.; red blood cells, 3,190,000.

Duodenal contents: Color, yellow. Urobilin, 400; urobilinogen, trace; total, 400+.

Tuberculosis. CASE 13 (162671).—Woman, aged thirty years. Tuberculous salpingitis. Tuberculous peritonitis found at operation elsewhere. Moderate splenomegaly. Hemoglobin, 35 per cent.; red blood cells, 4,410,000.

Duodenal contents: Color, brown. Bilirubin, ++. Urobilin, 400; urobilinogen, 600; total, 1000.

Chronic Septic Splenomegaly. CASE 14 (154572).—Woman, aged thirty-one years. Chronic septic splenomegaly. History of scarlet fever, frequent sore throat and "la grippe." Attacks of left upper abdominal pain two years. Cesarean section fifteen months previously. Two weeks afterward excruciating upper abdominal pain. Exploration elsewhere; large spleen found but nothing done. History suggestive of abdominal thrombophlebitis. Splenectomy April 12, 1916; spleen, 365 grams. Multiple infarcts. Hemoglobin, 50 per cent.; red blood cells, 4,600,000; white blood cells, 10,400.

Duodenal contents: Color, yellow. Urobilin, 400; urobilinogen, 0; total, 400.

Splenic Anemia. CASE 15 (158085).—Man, aged twenty-nine years. Splenic anemia. History of severe hemorrhages. Melena. Splenectomy; spleen, 780 (?) grams; liver moderately enlarged. Hemoglobin, 45 per cent.; red blood cells, 3,330,000; white blood cells, 3600.

Duodenal contents before splenectomy: Color, yellow. Bilirubin, trace. Urobilin, 1000; urobilinogen, 200; total, 1200.

Portal Atrophic Cirrhosis of Liver. CASE 16 (148570).—Man, aged sixty-two years. Portal atrophic cirrhosis of the liver. History of alcoholism. Ascites. Liver, 720 grams; spleen, 450 grams. Hemoglobin, 70 per cent.; red blood cells, 3,530,000.

Duodenal contents: Color, yellow. Bilirubin, ++. Urobilin, 1000; urobilinogen, trace; total, 1000+.

Polycythemia. CASE 17 (174186).—Man, aged fifty-one years. Polycythemia. Cyanosis. Liver moderately enlarged; spleen moderately enlarged. Diabetes, ten-year history. Nine months previously red blood cell count elsewhere, 9,500,000. Roentgen-ray treatment with improvement. At present hemoglobin, 93 per cent.; red blood cells, 5,320,000. Coagulation time ten minutes (Boggs). Bleeding time, five minutes.

Duodenal contents: Color, brown. Bilirubin, +++. Urobilin, 500; urobilinogen, 500; total, 1000.

Myelogenous Leukemia. CASE 18 (157746).—Man, aged thirty-two years. Spleen enormously enlarged; liver slightly enlarged. Slight degree of jaundice. Duration of history, one and a half years. Splenomegaly one year. Hemoglobin, 45 per cent.; red blood cells, 3,120,000; white blood cells, 496,000; myelocytes, 43.7 per cent.

Duodenal contents: Color, yellow. Urobilin, trace; urobilinogen, trace; total less than 200.

CASE 19 (158647).—Man, aged fifty-two years. Spleen enormously enlarged. Liver normal. Jaundice questionable. Length of history, one year. Splenomegaly, six months. Hemoglobin, 55 per cent.; red blood cells, 3,450,000; white blood cells, 307,000; myelocytes, 29.7 per cent.

Duodenal contents: Color, yellow. Bilirubin, trace. Urobilin, 2000; urobilinogen, 540; total, 2540.

CASE 20 (159989).—Man, aged thirty-nine years. Spleen moderately enlarged; liver normal. Jaundice? Length of history, twenty-two months. Splenomegaly, ten months. Hemoglobin, 53 per cent.; red blood cells, 3,210,000; white blood cells, 341,000; myelocytes, 29 per cent.

Duodenal contents: Color, yellow. Urobilin, 800; urobilinogen, 0; total, 800.

CASE 21 (173747).—Man, aged thirty-nine years. Spleen enormously enlarged; liver moderately enlarged. Slight jaundice. Splenomegaly, two years. Hemoglobin, 42 per cent.; red blood cells, 2,180,000; white blood cells, 8200; myelocytes, 15.7 per cent.

Duodenal contents: Color, brown. Bilirubin, +++. Urobilin, 900; urobilinogen, 200; total, 1100.

Combined Sclerosis. CASE 22 (154069).—Man, aged thirty-eight years. Very ataxic; duration six months. Neurological examination showed findings of advanced combined sclerosis. Wassermann tests negative. Hemoglobin, 54 per cent.; red blood cells, 3,720,000; color index, 0.7. The existence of pernicious anemia is highly probable.

Duodenal contents: Color, brown. Bilirubin, +++. Urobilin, 5500; urobilinogen, 2000; total, 7500.

A summary of the values is given in Table 1.

TABLE 1.—MISCELLANEOUS CASES: SUMMARY OF VALUES.

	Cases.	Urobilin.	Urobilinogen.
Anemia from hemorrhage (averages)	5	275+	0 or trace
Chronic arthritis (averages)	2	700	300
Dental abscesses	1	600	0
Cholelithiasis	1	600	0
Syphilis (averages)	2	900	0 or trace
Carcinoma	1	400	Trace
Tuberculous salpingitis	1	400	600
Chronic septic splenomegaly	1	400	0
Splenic anemia	1	1000	200
Portal atrophic cirrhosis	1	1000	Trace
Polycythemia	1	500	500
Myelogenous leukemia (averages)	4	925+	185
Combined sclerosis (pernicious anemia?)	1	5500	2000

All of these patients save 3 suffered from a moderate or severe anemia. The patient with polycythemia had a hemoglobin of 93

per cent. and a red cell count of 5,320,000 at the time of examination; the value for urobilin was 500 units, and that for urobilinogen 500 units. The patient with portal cirrhosis had a hemoglobin of 70 per cent. and a red cell count of 3,530,000; the urobilin was 1000 units and urobilinogen a trace. One of the patients with chronic infectious arthritis had a hemoglobin of 72 per cent. and a red cell count of 4,620,000; the urobilin was 800 units and urobilinogen zero. These three determinations are unaffected by anemia. They all show total values of approximately 1000 units. It has been concluded by other observers that total values of 1000 units or less are normal.

The values in cases of anemia from hemorrhage were especially low, possibly indicating an actual decrease of blood destruction below normal. In anemias of infectious origin, in syphilis, in carcinoma, and in cirrhosis of the liver the total values for urobilin and urobilinogen were 1000 units or less. Three of 4 patients with myelogenous leukemia gave low determinations; in 1, however, the total was 2540 units.

The group, as a whole, demonstrates consistently low values for the purely secondary types of anemia, irrespective of the severity of the anemia. Patients with simple anemia from hemorrhage present the lowest values of the series.

2. Hemolytic Jaundice.

Twelve determinations of the pigments in the duodenal contents have been made in 6 patients with hemolytic jaundice. Brief protocols of these cases follow:

Case 1 (112836).—Woman, aged forty-nine years. Acquired type of hemolytic jaundice, with the blood picture of a primary anemia. Increased fragility of erythrocytes. Gall-stones and a large slightly cirrhotic liver were found at operation. Weight of spleen, 910 grams. Patient returned during relapse one year and eight and a half months after splenectomy. The liver was then very large. Hemoglobin, 45 per cent.; red blood cells, 3,260,000; white blood cells, 3400; normoblasts, 152 in 300 cells. Duodenal test one year, eight and a half months after splenectomy: Color, brown. Bilirubin, +++. Urobilin, 3000; urobilinogen, 1000; total, 4000. The patient improved markedly after two transfusions, but a subsequent estimation of the pigments was not made.

Case 2 (153245).—Woman, aged thirty-eight years. Severe case of congenital type, with enlarged liver and gall-stones. Increased fragility of erythrocytes. Weight of spleen, 1700 grams. Hemoglobin, 47 per cent.; red blood cells, 2,840,000; white blood cells, 9800; no normoblasts.

Duodenal test one day before splenectomy: Color, yellow. Bilirubin, +++. Urobilin, 4600; urobilinogen, 1000; total, 5600.

Thirty-eight days after splenectomy: Color, yellow. Bilirubin, trace. Urobilin, 1400; urobilinogen, 1800; total, 3200. The hemoglobin at the time of the latter test was 70 per cent.; red cells, 4,960,000; no normoblasts.

Case 3 (148209).—Man, aged twenty years. Mild case of congenital type. Increased fragility of erythrocytes. At operation the liver showed evidence of early cirrhosis. Weight of spleen, 300 grams. Hemoglobin, 70 per cent.; red blood cells, 4,920,000.

Duodenal test forty-seven days before splenectomy: Color, yellow. Bilirubin, trace. Urobilin, 1400; urobilinogen, 1000; total, 2400. Fourteen days before splenectomy: Color, yellow. Bilirubin, trace. Urobilin, 2000; urobilinogen, 1200; total, 3200. Thirteen days after splenectomy: Color, yellow. Urobilin, 800; urobilinogen, 1000; total, 1800. One hundred and forty days after operation: Color, yellow. Urobilin, trace; urobilinogen, 400; total, 400+.

Case 4 (161538).—Woman, aged twenty-seven years. Moderately severe case of congenital type. Increased fragility of erythrocytes. Liver probably normal. Gall-stones present. Weight of spleen, 560 grams. Hemoglobin, 64 per cent.; red blood cells, 3,860,000.

Duodenal test three days before splenectomy: Color, yellow. Urobilin, 500; urobilinogen, 500; total, 1000. Twenty-three days after splenectomy: Color, light brown. Bilirubin, trace. Urobilin, 400; urobilinogen, 0; total, 400. At the time of the latter test the hemoglobin was 70 per cent.; red blood cells, 4,680,000.

Case 5 (162670).—Man, aged thirty-one years. Moderately severe case, probably of congenital type. Increased fragility of erythrocytes. Liver normal. Gall-stones present. Spleen weighed 1250 grams. Hemoglobin 67 per cent.; red blood cells, 3,650,000. Considerable deformity of red cells.

Duodenal test four days before splenectomy: Color, brown. Bilirubin, +++. Urobilin, 1400; urobilinogen, 1800; total, 3200. Twenty-one days after splenectomy: Color, brown. Bilirubin, ++. Urobilin, 1000; urobilinogen, 200; total, 1200. The hemoglobin at the time of the latter test was 80 per cent.; red blood cells, 4,022,000.

Case 6 (153653).—Man, aged twenty-three years. Mild case with some evidence that a familial factor was present. Increased fragility of erythrocytes. Non-operative. Spleen moderately enlarged. Hemoglobin, 60 per cent.; red blood cells, 3,900,000; white blood cells, 5800; no normoblasts.

Duodenal test: Color, dark yellow. Bilirubin, +. Urobilin, 3000; urobilinogen, trace; total, 3000+. (The low urobilinogen may have been due to delay in making the estimation.)

The values for urobilin and urobilinogen in the duodenal contents are very markedly increased in cases of hemolytic jaundice. A few reported cases have shown even higher values than those we have

demonstrated. High values are found even when a moderate degree of anemia is present. In Case 3 at the time of the first two tests the anemia was not severe; however, abnormally large amounts of pigment were obtained. Severe grades of anemia are associated with very high values; in Case 2, with a red count of 2,840,000, the total values were 5600 units. If these values are a reasonably accurate index of hemolysis, blood destruction in hemolytic jaundice is probably much increased at a time when blood production is not seriously affected. This is in contrast to our experience with pernicious anemia in which the evidence of bone-marrow insufficiency is usually marked and the evidence of blood destruction extremely variable. Patients with pernicious anemia who show high values for urobilin and urobilinogen at a time when the blood count is low frequently show very low values when the blood count has risen to the level of a moderate anemia. Patients with hemolytic jaundice, on the other hand, may show high values with only a slight anemia. In 2 patients with very high values the blood picture simulated that of pernicious anemia. An excessive degree of blood destruction probably exhausted the bone marrow.

In 4 of these patients tested before and after operation there was an appreciable reduction in the values for bile pigments following splenectomy. In 2 of them a decided decrease in the amount of urobilinogen was revealed. In Case 3 a former operation for cholecystitis had not reduced the values to normal. The preoperative values of these 4 patients averaged 2050 units for urobilin and 1100 units for urobilinogen, a total of 3150 units. The average postoperative values at periods varying from thirteen days to four months after splenectomy were 800 units for urobilin and 625 units for urobilinogen, a total of 1425.

TABLE 2.—ESTIMATION OF PIGMENTS IN THE DUODENAL CONTENTS. HEMOLYTIC JAUNDICE.

Case No.	Time before and after splenectomy.	Color.	Bilirubin, units.	Urobilin, units.	Urobilinogen, units.	Total, units.
1 (112836)	1 year 8½ months after	Brown	+++	3000	1000	4000
2 (153245)	1 day before	....	+++	4600	1000	5600
	38 days after	Yellow	Trace	1400	1800	3200
3 (148209)	47 days before	Yellow	Trace	1400	1000	2400
	14 days before	Yellow	Trace	2000	1200	3200
	13 days after	Yellow	0	800	1000	1800
	140 days after	Yellow	0	Trace	400	400+
4 (161538)	3 days before	Yellow	....	500	500	1000
	23 days after	L. brown	Trace	400	0	400
5 (162670)	4 days before	Brown	+++	1400	1800	3200
	21 days after	Brown	++	1000	200	1200
6 (153653)	Non-operative	D. yellow	+	3000	Trace	3000+
Average values before splenectomy			4 cases	2050	1100	3150
Average values after splenectomy			4 cases	800	675	1475

3. Pernicious Anemia.

Eighty-five estimations were made in 61 cases of pernicious anemia. In 6 of the cases the tests were done only after splenectomy, thereby reducing the number of medical and preoperative observations from 61 cases to 55 cases. The average of the readings for urobilin in these 55 cases was 1856.5 units and that for urobilinogen 1604.5 units. The average total for urobilin and urobilinogen was therefore 3461 units. This average is approximately four times normal and remarkably close to the figure obtained by Schneider in his recently reported series.

Nine of the 55 patients showed total values less than 1000; in other words, 84 per cent. gave values of 1000 units and over. It is our experience that patients more than fifty-five years of age, and particularly more than sixty years of age, not uncommonly show low values. In these senile types there is frequently evidence of advanced bone-marrow damage with little active hemolysis. Some of these anemias may, in reality, be osteosclerotic in origin, but this distinction is difficult to make clinically.

It is also our experience that as the blood improves in pernicious anemia the duodenal values quickly decline. When the red cells reach 3,500,000 the duodenal values are quite apt to run below 1000 units total. The average in 3 patients with red cell counts above 3,500,000 cells was 433.3 units for urobilin and 466.6 units for urobilinogen, making a total of 899.9 units. Moreover, a few patients who have had repeated tests during treatment have shown this same rapid decline in values when the anemia became of moderate grade. Hemolytic jaundice, on the other hand, gave high values even when the anemia was slight.

It is therefore to be concluded that a certain number of patients with undoubted pernicious anemia do show low total values at certain times. On the other hand, urobilinogen will be present in an appreciable amount. The most noteworthy constant is the presence of an estimable amount of urobilinogen. Urobilinogen was absent in only 1 of the 55 medical and preoperative cases; in 2 others there was a trace; in 52 urobilinogen was present in relatively large amounts; in 24, or nearly one-half of the cases, urobilinogen was present in even larger amounts than urobilin. The presence of large quantities of urobilinogen before splenectomy and its complete absence in at least 75 per cent. of the cases after splenectomy are very striking findings and may have an important significance.

The highest total values were obtained in patients with red cell counts between 2.5 and 3.5 million cells. They were slightly lower in patients with erythrocyte counts between 1.5 and 2.5 million cells and considerably lower in patients with erythrocyte counts below 1.5 million cells. The lowest values were obtained in patients

with counts over 3.5 million cells, but even in these urobilinogen was present in a distinctly abnormal amount. The most active hemolysis seems to occur in the patients with counts between 2.5 and 3.5 million cells, but this may be due to the fact that patients are only rarely seen at the onset of a period of active blood destruction. As a group these latter cases show evidence of very active hemolysis and at the same time evidence of active blood production.

TABLE 3.—ESTIMATION OF PIGMENTS IN THE DUODENAL CONTENTS: VALUES WITH RESPECT TO ERYTHROCYTE COUNT.

PERNICIOUS ANEMIA (MEDICAL AND PREOPERATIVE).

	Number of estimations.	Average urobilin, units.	Average urobilinogen, units.	Total, units.
Erythrocytes 1.5 million and below	11	1496.3	1472.7	2969.0
Erythrocytes 1.5 to 2.5 million .	32	1864.3	1425.0	3589.3
Erythrocytes 2.5 to 3.5 million .	13	2346.1	1653.8	3999.9
Erythrocytes 3.5 million and over	3	433.3	466.6	899.9
HEMOLYTIC JAUNDICE (MEDICAL AND PREOPERATIVE.)				
Erythrocytes 3.5 million and over	4	1650.0	850.0	2500.0

Age. The average totals for patients under fifty-five years of age showed very little variation by decades. Over the age of fifty-five years there was a decided drop both in urobilin and urobilinogen. Between the ages of fifty-six and sixty years the totals average 2644.2; over the age of sixty years they average 1600, while under fifty-five years the average totals for decades vary between 3325 and 4238.

TABLE 4.—ESTIMATION OF PIGMENTS IN THE DUODENAL CONTENTS: VALUES WITH RESPECT TO AGE.

PERNICIOUS ANEMIA (MEDICAL AND PREOPERATIVE).

Age of patient.	Number of cases.	Average urobilin, units.	Average urobilinogen, units.	Total, units.
30 years and under .	3	2266.6	1833.3	4099.9
31 to 40	10	1955.0	1370.0	3325.0
41 to 50	12	2075.0	1691.6	3766.6
51 to 55	16	2200.6	2037.5	4238.1
56 to 60	9	1372.0	1272.2	2644.2
Over 60 years . .	5	660.0	940.0	1600.0

Size of Spleen. High values were obtained both in patients with large spleens and in those with small spleens. Lower values were obtained in those with spleens of moderate size, that is, weighing from 200 to 500 gm. The highest urobilinogen values were obtained in patients with spleens of 200 gm. and less. It is impos-

sible to determine the significance of these findings with respect to the size of the spleen. The degree of pathological or of functional damage in the liver may be the important factor. Patients with small spleens have more constantly shown evidence of advanced pathological change in the liver.

TABLE 5.—ESTIMATION OF PIGMENTS IN THE DUODENAL CONTENTS: VALUES WITH RESPECT TO WEIGHT OF SPLEEN.

PERNICIOUS ANEMIA (PREOPERATIVE ESTIMATIONS).

Weight of spleen.	Number of cases.	Average urobilin, units.	Average urobilinogen, units.	Total, units.
200 grams and below .	4	3000.0	2000.0	5000.0
200 to 500 grams . .	12	2342.5	1487.5	3830.0
Over 500 grams . .	6	3233.3	1433.3	4666.6

Values after Splenectomy.

A very striking reduction is observed in the values after splenectomy. A total of 19 patients was examined after splenectomy; 13 both before and after operation. The average of the total values for these 13 patients became reduced from 4492.2 to 1134.6 units. Urobilinogen in 10 of the 13 cases was reduced to zero or a trace. Of the entire group of 19 examined after splenectomy, 13 gave values for urobilinogen of zero or a trace. Three showed urobilinogen over 1000 units after splenectomy, but judging from the clinical history these readings probably became reduced later.

Two patients who had high values for urobilinogen following splenectomy, showed no definite improvement in the anemia. The liver was enlarged in each instance and an exposure of the liver to radium was suggested. The application of 50 mg. of radium over five areas for a total of ten hours was followed by a very marked reduction in the size of the liver, a fall in duodenal values and a prompt improvement in the anemia.

TABLE 6.—ESTIMATION OF PIGMENTS IN THE DUODENAL CONTENTS: PREOPERATIVE AND POSTOPERATIVE VALUES.

	Number of cases.	Average urobilin, units.	Average urobilinogen, units.	Total, units.
Pernicious anemia (medical and preoperative)	55	1856.5	1604.5	3461.0
Pernicious anemia (preoperative) .	13	2970.7	1521.5	4492.2
Pernicious anemia (postoperative)	13	815.4	319.2	1134.6
Pernicious anemia (postoperative)	10 of 13	480.0	0 or trace	480.0+
Hemolytic jaundice (preoperative)	4	2050.0	1100.0	3150.0
Hemolytic jaundice (postoperative)	4	800.0	675.0	1475.0

TABLE 7.—ESTIMATION OF PIGMENTS IN THE DUODENAL CONTENTS. PERNICIOUS ANEMIA (SPLENECTOMY).

Case No.	Date of splenectomy.	Time before and after splenectomy.	Color.	Bilirubin, units.		Urobilin, units.		Urobilinogen, units.		Total, units.		Red blood cells, millions.	Weight of spleen, grams.
				Before operation.	After operation.	Before operation.	After operation.	Before operation.	After operation.	Before operation.	After operation.		
1 (152922)	Mar. 10, 1916	7 days before	...	+++	...	2000	...	600	...	2600	...	1.96	150
		21 days after	Yellow	...	+	...	1400	...	0	...	1400	1.48	
2 (152671)	15, 1916	20 days before	...	...	...	5000	...	1000	...	6000	...	2.94	508
		13 days before	...	...	...	6000	...	4000	...	10000	...		
		21 days after	...	...	...	...	800	...	0	...	800	4.40	
3 (154493)	25, 1916	9 days before	Brown	+	...	3000	...	2000	...	5000	...	2.34	525
		17 days after	Yellow	...	++	...	200	...	0	...	200	1.89	
4 (153395)	29, 1916	9 days before	Brown	+++	...	3500	...	Trace	...	3500+	...	2.53	450
		24 days after	Yellow	...	Trace	...	Trace	...	Trace	...	200−	3.14	
5 (154299)	April 8, 1916	9 days before	Yellow	+++	...	4000	...	2000	...	6000	...	2.80	133
		21 days after	Brown	...	++	...	6800	...	1400	...	8200	2.73	
		4 mos. after	Brown	...	+++	1800	...	1400	...	3200	...	2.16	
6 (157146)	20, 1916	3 days before	Brown	+++	...	3200	...	2600	...	5800	...	2.96	270
		19 days after	Brown	...	...	...	1400	...	Trace	...	1400+	3.25	
7 (151021)	May 4, 1916	5 days before	Yellow	...	...	5400	...	4600	...	10000	...	2.46	111
		27 days after	Yellow	...	Trace	...	200	...	0	...	200	1.96	
8 (157663)	June 2, 1916	1 mo. before	Brown	+	...	840	...	760	...	1600	...	2.41	410
		14 days before	Yellow	...	...	500	...	600	...	1100	...	2.22	
		2 mos. after	Yellow	...	+	...	400	...	0	...	400	3.49	
9 (157290)	5, 1916	2 mos. before	Yellow	...	...	600	...	800	...	1400	...	1.66	192
		18 days after	Yellow	...	Trace	...	Trace	...	Trace	...	200−	5.20	
10 (161677)	15, 1916	6 days before	Yellow	+	...	400	...	400	...	800	...	2.72	770
		36 days after	Yellow	...	++	...	Trace	...	0	...	200−	3.00	
11 (160970)	27, 1916	11 days before	Brown	+++	...	800	...	200	...	1000	...	3.33	300
		29 days after	Yellow	...	Trace	...	Trace	...	0	...	200−	4.38	
12 (158136)	28, 1916	2 mos. before	Brown	+++	...	2500	...	2000	...	4500	...	1.51	300
		7 days before	Brown	+++	...	600	...	600	...	1200	...	1.94	
		23 days after	Yellow	...	++	...	800	...	1400	...	2200	1.80	
13 (170115)	Oct. 3, 1916	8 days before	Brown	+++	...	8000	...	2000	...	10000	...	2.35	600
		1 mo. after	Brown	...	+++	...	800	...	1300	...	2100	2.65	
		2 mos. after	Yellow	...	+	...	600	...	800	...	1400	3.14	

Transfusions.

Transfusions did not seem to affect the duodenal values. However, estimations were not made the first or second day following transfusion, at which time an increase in the amounts of pigments might be expected. Estimations made five and ten days following transfusion gave no unusual values.

Bilirubin.

An excess of bilirubin was usually present when large amounts of urobilin and urobilinogen were found. This relationship was by no means constant. An excess of bilirubin was not infrequently found with low values for urobilin and urobilinogen, and very small amounts of bilirubin were occasionally found with high values. The amount of bilirubin therefore does not run parallel with the values for urobilin and urobilinogen. These values are probably indicative of some quite different function or impairment of function in pathological conditions.

Summary.

1. With a few slight modifications of technic we have used the method of Schneider in estimating quantitatively the amounts of urobilinogen and urobilin in the duodenal contents obtained by means of an Einhorn tube. The procedures are simple and can be carried out in any clinical laboratory. The results are comparable with those obtained by the more complicated and time-consuming methods in which stool extracts are used for the estimation of these pigments.

2. In a group of 22 miscellaneous cases, low values were obtained in patients with anemia from hemorrhage, carcinoma, tuberculous peritonitis, syphilis, portal cirrhosis, chronic infectious arthritis, and gall-stones. They were low in 3 of 4 patients with myelocytic leukemia. The amounts of these pigments were especially low in cases of anemia from hemorrhage.

3. In hemolytic jaundice the values were consistently high even when severe anemia was not present. The values fell appreciably after splenectomy, but not as promptly as in pernicious anemia.

4. In pernicious anemia the amounts of urobilin and urobilinogen in the duodenal contents were above normal in 84 per cent. of the cases. The amount of urobilinogen was constantly increased when the anemia was severe. Patients over the age of fifty-five showed lower values than younger patients. The values presented no definite relationship to the size of the spleen. Following splenectomy there was a very definite decrease in the amounts of urobilin and urobilinogen; the decrease in urobilinogen was especially noticeable.

5. The amounts of bilirubin in the duodenal contents did not run constantly parallel to the amounts of urobilin and urobilinogen.

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PLEURAL EOSINOPHILIA.

By Rene Bine, M.D.,

ASSISTANT IN MEDICINE, UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL, SAN FRANCISCO.

Reviewing 2758 articles for his monograph on eosinophilia, E. Schwarz,[1] in 1914, was able to find but 68 cases of pleural eosinophilia, although this condition had been described by Widal and Ravaut[2] as early as 1900. Bayne-Jones[3] believes the phenomenon occurs more frequently than this would seem to indicate, and attributes the small number of reported cases to the fact that many pleural fluids have not been examined microscopically and that relatively few have been stained with polychrome dyes. According to Bayne-Jones, pleural eosinophilia occurs in about 1 to 5 per cent. of all cases of pleural effusion, as determined by investigators applying constant methods to cases in series.

In many cases of primary pleurisy, when the fluid is examined on the first day of its appearance, a very slight, transient eosinophilia is noted.[4] This eosinophilia vanishes rapidly before the onrush of the neutrophiles. Again during convalescence, eosinophiles may be encountered in small numbers. In these cases, however, the percentage of eosinophiles seldom reaches and does not exceed 5 per cent., in marked contrast to the so-called group of true pleural eosinophilias where these cells range from 10 per cent. upward, and

[1] Die Lehre von der allgemeinen und örtlichen "Eosinophilie," Erbeg. d. allg. Path. u. path. Anat., 1914, xvii, 138–790.
[2] Applications cliniques de l'étude histologique des epauchements sero-fibrineux de la plèvre, Compt. rend. Soc. de biol., 1900, 648.
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[4] Malloizel: Recherches anatomo-cliniques sur les reactions pleurocorticales, Thèse de Paris, 1907.

are, in addition, of rather early appearance and far greater persistence. Bayne-Jones's figures[5] as to incidence probably include the relative as well as the true pleural eosinophilias.

The etiology of pleural eosinophilia is purely a matter of speculation. It has been found in connection with such a varied pathology as to be devoid of diagnostic significance. With the exception of one patient with gonorrheal sepsis[6] in no case has the causal germ been one in whose products of inflammation eosinophilia is ordinarily encountered. Among the other conditions with which it has been found are trauma, tuberculosis, sepsis, typhoid fever, syphilis, pleuropericarditis, pneumonia, polyarthritis, nephritis, pulmonary gangrene, hemorrhagic infarct of the lung, endothelioma, septic endocarditis, myocarditis, cardiac insufficiency, puerperal sepsis, neoplasm, and influenza. In practically all of the cases the effusions have been comparatively small, and most have been sterile.

The local often precedes the general eosinophilia and usually exceeds it in degree. In most instances the local is first to disappear. This has been interpreted by some as pointing to a local production of eosinophiles. Others explain it as follows: The local accumulation of cells occurs in response to the irritation of definite eosinotactic substances, probably protein split products in the exudate undergoing absorption. If their field of action extends to the bone marrow the blood eosinophilia follows. If the irritability of the hematopoietic system persists longer than the local eosinotactic stimulus we find the general outlasting the local eosinophilia.

Attention has been called to the small number of cases associated with tuberculosis, the most frequent cause of pleural effusion. This, however, is not surprising. In tuberculosis the pleural involvement is usually of slow, insidious onset and the absorption of the effusion is correspondingly slow and gradual. In most cases of tuberculosis we find a decrease of the eosinophiles in the blood, this decrease becoming more pronounced as the disease approaches a fatal termination and the patient loses his power of reaction. On the other hand, tuberculin injections have been shown to produce at least transient eosinophilias in cases not too far advanced, and attempts have been made to use this reaction as a guide to treatment. It is probable that in the cases with pleural effusion there are ordinarily not sufficient protein split products liberated to produce an eosinophilic reaction. In addition to this, we must remember that in tuberculosis the pleura is often thickened, adhesions frequently walling off and confining the effusion, and that a thickened pleura or fibrinous deposits naturally offer greater barriers to the absorption of protein split products present in the effusion.

The following case was recently reported by Petzetakis:[7] In the

[5] Bayne-Jones: Loc cit.

[6] Jacob, F. H.: A case of Gonococcal Pyemia, British Med. Jour., 1907, xii, 203.

[7] Reactions pleurales parabronchitiques, Bull. et mém. Soc. med. d. hôp. de Paris, 1916, xl, 1545.

course of an acute bronchitis very slight bilateral effusions were discovered, possibly only because the author was studying so-called "parabronchitic pleural reactions." The right-sided effusion was found on the first day of observation. It contained no eosinophiles. On the third day there were 4 per cent.; on the fifth, 10 per cent.; whereas on the left side there were none. On the seventh day no fluid could be obtained from the left side; the right contained 41 per cent. eosinophiles; seven days later but 15 per cent. The blood showed 1 per cent. on the third day, 1.5 per cent. on the fifth, 2 per cent. on the seventh, and 8 per cent. on the ninth day.

Bayne-Jones has analyzed the 8 cases of eosinophilic pleurisy previously collected by Schwarz and has added 1 case of his own to the list. I have had 2 cases of pleural eosinophilia under observation, presenting rather unusual clinical features.

Case I.—A. A.; white; aged twenty-nine years; seen at the French Hospital in consultation with Dr. Paul Campiche.

The patient was a cook, born in Switzerland; had been in Alaska; never visited the tropics. He had never been ill before the onset of his present trouble. Six months previously he had been found in perfect health and was admitted to a Swiss Relief Society.

His present illness began with cough, pain in the anterior chest, vomiting, and diarrhea. The symptoms lasted six days before hospital admission.

Physical examination showed a well-developed and well-nourished man, with a temperature of 102°, and a weak, regular, possibly dicrotic pulse. The face was flushed. The patient looked anemic. The area of cardiac dulness was not increased; a loud, systolic murmur was audible in mitral and pulmonic areas. The chest showed a slight fulness at the right base posteriorly, with slight dulness at the right base and right axilla, with bronchial breathing and a few small moist rales. The abdomen was tympanitic, with no tenderness, tumors, or roseola. The liver and spleen were not enlarged.

The temperature chart, in connection with the above symptoms and signs, led to a diagnosis of an initial pneumonia, in which the crisis had taken place and been succeeded by a pleural exudate which possibly had become purulent. The general condition and the history of diarrhea suggested the possibility of typhoid or miliary tuberculosis, though the fever curve was rather that of a septic process. A needle was introduced in the dull area at the right base, but no fluid was obtained. Urine showed albumin and a positive diazo. The white blood cells were 8200, with polynuclear neutrophiles, 74 per cent.; lymphocytes, 25 per cent.; large mononuclears, 1 per cent.; no eosinophiles; no plasmodia. One stool consisted of almost pure bright red blood, with no mucus, amebæ, parasites, or eggs. Sputum was watery, with traces of blood; just enough obtained to make one smear; few cocci seen; no tubercle bacilli.

Widal reaction was negative at end of forty minutes, with dilution 1 to 40, and two different cultures.

The bloody stool, the first and only one during the course of the disease, suggested an embolic process, though with the leukopenia and positive diazo; typhoid was not absolutely ruled out until a week later, when the Widal reaction was again found negative in all dilutions and blood culture still showed no growth.

Radiographs taken one week later showed a well-defined shadow at right base, extending upward to the scapular spine, and also a mediastinal shadow. After repeated needle punctures on successive days, on one occasion a little serous fluid was obtained. Smears showed numerous leukocytes; no bacteria; no tubercle bacilli.

Ten days after onset the patient developed a thrombosis of the left popliteal and saphenous veins, and showed evidence of a partial pneumothorax. Blood count now was as follows: Hemoglobin, 50 per cent.; red blood cells, 3,500,000; white blood cells, 8000; polynuclears, 74 per cent.; large mononuclears, 4 per cent.; lymphocytes, 18 per cent.; eosinophiles, 0. Wassermann, blood culture, and von Pirquet were negative.

One month after admission radiographs showed: (1) a definite shadow on right side, from about the level of the spine to the angle of the scapula; (2) a shadow as if the dome of the diaphragm were pushed up; (3) large dilated heart; (4) increased shadow of great vessels. Blood count at this time gave: white blood cells, 7600; polynuclears, 74 per cent.; mononuclears, 6 per cent.; lymphocytes, 19 per cent.; eosinophiles, 1 per cent. Widal negative.

Needle puncture made in scapular line right side, ninth space, yielded a yellowish-green serous fluid, which microscopically showed a large number of white blood cells, no red blood cells. Differential count: Polynuclears, 14 per cent.; large mononuclears, 5 per cent.; lymphocytes, 30 per cent.; eosinophiles, 51 per cent. Apparently the upper focus was primary and the effusion secondary to it.

Four days later complement-fixation reactions for syphilis and echinococcus were negative. White blood cells, 4000; polynuclears, 73 per cent.; mononuclears, 4 per cent.; lymphocytes, 22 per cent.; eosinophiles, 1 per cent.

Recovery was steadily progressive and the patient left the hospital three months after admission.

He was next seen four years later, when examination showed that the right lung moved less than the left, but was within normal limits. There were no abnormalities on percussion or auscultation. The right border of the heart extended 1 cm. to the right of the sternum; left border to midclavicular line; sounds were normal. Patient was much stouter than when last seen. Blood: Hemoglobin, 75 per cent.; red blood cells, 4,800,000; white blood cells, 6400; polynuclear neutrophiles, 55 per cent.; lymphocytes, 12 per cent., polynuclear eosinophiles, 32 per cent.; basophiles, 1 per cent.

CASE II.—F. A.; white; male; aged thirty-seven years (Hosp. No. 10697); admitted to the University of California Hospital on January 26, 1916, in the service of Dr. Herbert C. Moffitt. Born in Portugal; had lived in Brazil, age fourteen to twenty-one, working as a farmer on a coffee plantation; in California, age twenty-one to thirty-seven, working in Isleton, on the Sacramento River, raising asparagus.

Family history, habits, and venereal history were unimportant. He had malaria for one month when sixteen and again for two weeks at thirty-one; cured by quinin. In South America had spells of diarrhea, one or two yearly, lasting from three to seven days; no blood.

Present Illness. Was well until January 1, 1916.

January 2 he tried to lift a heavy heap of hay and strained his back.

January 3 began to have four or five thin, watery stools daily, with stringy material but no blood. Diarrhea lasted one week. After cessation of diarrhea he began to have dull, frontal headaches and general malaise. Headaches have persisted, but now has no abdominal pain or diarrhea.

January 20 his hearing became impaired and has steadily grown worse.

January 22 he noticed edema of feet, and this has increased to the present. Feels weak and walks with pain. No vomiting or nausea. For the past five days has had increasing cough, with sputum, and for the past two days has had a pain in the left lower thorax, increased by respiration and cough.

Examination. Very sick looking; flushed cheeks; respiration rapid; frequent cough. Herpes labialis. Heart very rapid, but not enlarged; sounds normal, except for very faint systolic, probably functional murmur. Lungs: dulness, with absent fremitus and decreased breathing over the left base, not extending beyond the posterior axillary line. Slight Grocco on right side. Friction rub heard just outside the left nipple. Very tender over lower ribs in left axilla and in left upper quadrant of abdomen, but there is no muscle rigidity. Blood: Hemoglobin, 80 per cent.; red blood cells, 5,280,000; white blood cells, 12,000.

Diagnosis. Facies and history would suggest typhoid, but breathing and herpes point to a respiratory infection. There is so much tympany in front, on the left side, and such a marked absence of fremitus in the back, that, despite the moderate mobility of the side and the audible breath sounds, the patient probably has a moderate amount of fluid at the left base.

February 2 blood culture was sterile. Wassermann in serum negative. Exploratory puncture of pleural cavity: Needle inserted in line with angle of scapula in both ninth and tenth spaces, but no fluid obtained. Then inserted in ninth space in paravertebral line

and 50 c.c. of straw-colored, murky fluid withdrawn. Pleuritic fluid: fibrin clot settles out with standing. No organisms seen. Cytology: polymorphonuclears, 93 per cent.; endothelials, 4 per cent. lymphocytes, 3 per cent.; many eosinophiles, 30 to 40 per cent. Cultures made from fluid remained sterile. Urine: acid; heavy trace of albumin; urobilin; several hyaline, few granular casts; moderate red and white cells.

Two days later. Sputum cultures: Gram-negative coccus recovered and a few Gram-positive lanceolate diplococci. Sputum washed in normal salt and injected into peritoneal cavity of white mouse. Mouse died February 5, 1916. Smear from peritoneal cavity showed Gram-positive lanceolate diplococci. No Gram-negative cocci. Widal agglutination test: negative in all dilutions. Tuberculin skin test: very slightly positive to human and bovine. Blood: Hemoglobin, 82 per cent.; red blood cells, 5,300,000; white blood cells, 13,000. Polynuclear neutrophiles, 70 per cent.; lymphocytes, 7 per cent.; large mononuclears and transitionals, 6 per cent.; eosinophiles, 16.5 per cent.; basophiles, 0.5 per cent. (350 cells counted). Urine: neutral; trace of albumin; casts as above.

Spinal puncture: needle inserted in the fourth lumbar space. Fluid: clear; drops slowly; no increase of pressure; about 15 c.c. withdrawn. Nonne; no Noguchi; faint haze. Fehling's reduced. Cells: one lymphocyte to cubic millimeter. Wassermann in spinal fluid negative. No trichinæ found in centrifugalized sediment of fluid; 10 c.c. blood taken and laked with acetic acid. Specimen centrifugalized and examined for trichinæ; none found. Stools showed no parasites or ova.

February 1 patient had a short, hacking cough and brought up from 30 to 60 c.c. of heavily blood-stained sputum. Blood: neutrophiles, 64 per cent.; lymphocytes, 10.5 per cent.; large mononuclears and transitionals, 10.5 per cent.; eosinophiles, 14.5 per cent.; basophiles, 0.5 per cent. (300 cells counted).

February 4 fluoroscopic examination showed limitation of motion in left diaphragm, which is 1 cm. higher than the right. Movement of right diaphragm increased. Small, well-defined shadow at the left base, distinct from the heart, shading off gradually into the costal shadow above and not affected by change of position.

February 8 the blood was as follows: Hemoglobin, 90 per cent.; red blood cells, 5,350,000; white blood cells, 10,150; polynuclear neutrophiles, 68 per cent.; lymphocytes, 10 per cent.; large mononuclears and transitionals, 10 per cent.; eosinophiles, 10 per cent. (148 cells counted).

February 14 no cough, no sputa, and all pain and distress have gone. Temperature is normal. Lungs have cleared except for a little impaired resonance at bases. Blood: Hemoglobin, 80 per cent.; red blood cells, 5,872,000; white blood cells, 11,800; polynuclear

neutrophiles, 48 per cent.; lymphocytes, 12 per cent.; large mononuclears and transitionals, 20.5 per cent.; eosinophiles, 19 per cent.; basophiles, 0.5 per cent. Discharged well.

Both of these patients had atypical pulmonary infections, followed by pleural effusions. In the first case the blood showed 1 per cent. eosinophiles, the pleural fluid 51 per cent. Four years later, long after the patient's complete recovery, the blood still contained 32 per cent. eosinophiles. In the second case the pleural fluid contained 30 to 40 per cent. eosinophiles, the blood 16 per cent., then 14.5 per cent., 10 per cent., and 19 per cent., three weeks after the first count. In the reported cases the local eosinophilia precedes the general, is usually of a greater degree, and is the first to disappear. In our first case four years after the effusion the blood eosinophilia was 32 per cent., a count remarkable for its degree and long persistence. We have found no case recorded presenting these features.

To Dr. Campiche, who placed his patient in my care from the first, and to Dr. Moffitt for the privilege of reporting Case II, I herewith express my appreciation.

WAR MEDICINE

CONTRIBUTORS.

MAJOR SIMON FLEXNER, M.R.C., U. S. A. (Director, Rockefeller Institute).
DR. A. E. TAYLOR, U. S. Food Administration (Prof. of Physiological Chemistry, Univ. of Penna.).
ASSISTANT SURGEON-GENERAL J. W. TRASK, U.S.P.H.S.
ASSISTANT SURGEON-GENERAL W. C. RUCKER, U.S.P.H.S.
COLONEL FREDERICK F. RUSSELL, M.C., U. S. A.
DR. RICHARD M. PEARCE, National Research Council (Prof. of Research Medicine, Univ. of Penna.).
LIEUTENANT-COLONEL E. G. BRACKETT, U. S. N. A.
MAJOR R. M. YERKES, Sanitary Corps, U. S. A. (Prof. of Psychology, Harvard University).
SURGEON M. J. ROSENAU, U.S.N.R.F. (Prof. of Preventive Medicine and Hygiene, Harvard Medical School).
MAJOR R. TAIT MCKENZIE, R.A.M.C. (Prof. of Physical Therapeutics, Univ. of Penna.).
MR. DOUGLAS C. MCMURTRIE, Director of Red Cross Institute for Crippled and Disabled Men, New York.
DR. E. E. SOUTHARD, Director of Psychopathic Department, Boston State Hospital.
LIEUTENANT-COLONEL WARFIELD T. LONGCOPE, U. S. N. A. (Prof. Practice of Medicine, Columbia University).
DR. C. V. CHAPIN, Superintendent of Health, Providence, R. I.
DR. GRACE L. MEIGS, Children's Bureau, U. S. Department of Labor.
DR. A. N. RICHARDS, Professor of Pharmacology, Univ. of Penna.
DR. W. H. F. ADDISON, Assistant Professor of Histology, Univ. of Penna.
DR. DAVID L. EDSALL, Professor of Clinical Medicine, Harvard Medical School.
DR. GRAHAM LUSK, Professor of Physiology, Cornell Medical School.
DR. O. H. PERRY PEPPER, Associate in Medicine, Univ. of Penna.
DR. W. P. LUCAS, American Red Cross (Prof. of Pediatrics, Univ. of California).
MAJOR FRED H. ALBEE, M.R.C., Director of Military Reconstruction Hospital No. 3, U. S. A.
DR. RUFUS COLE, Director Rockefeller Hospital.
LIEUTENANT E. ESTELL LEE, M.R.C., U. S. A.
DR. JOHN A. HARTWELL, Assist. Prof. of Surgery and Prof. Clinical Surgery, Cornell Medical School.
DR. RALPH PEMBERTON, American Red Cross.
DR. DOROTHY REED MENDENHALL, Children's Bureau, U. S. Department of Labor.
ASSISTANT SURGEON PAUL A. LEWIS, U.S.N.R.F. (Pathologist to Phipps Institute, Philadelphia).

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With the entry of the United States into the War the June number of this magazine inaugurates a policy of systematic consideration of the important and enlarged aspects of the care of the cripple which war entails. These have, of course, long since necessitated attention in Europe. The *American Journal of Care for Cripples* should be well qualified for the work it proposes to do. Octavo in size, with page margins of three and four inches, presumably to allow of large, full-page illustrations, it has probably few rivals among magazines at large in substantial impressiveness. Indeed, in these war days wonder is legitimate whether such unusual margins are justifiable. The journal is in its fourth volume and is, of course, already well known to those interested in its aims under peace conditions. The reason for its review in these columns is the added interest of its enlarged scope.

The United States and the War Cripple. By way of introduction the editor says under this caption, in part: "The literature on this

subject (The War Cripples) is scanty and scattered, and much of it is available only in foreign languages. Even then most of the material cannot be found outside of libraries in the largest cities. For these reasons the journal will undertake responsibility for making available to its readers much of the significant and authoritative literature dealing with provision for war cripples. The foreign material will appear in English translation, on the literary form of which little emphasis will be laid. The content and information will be the main object of publication. Original contributions are usually valued to an artificial degree by high-standard publications. In a new subject, such as work for war cripples, however, insistence upon original articles would only cause delay and necessitate omission of much valuable matter. The journal will therefore pass on to its readers useful articles from whatever sources they may be obtained. Its best services can be rendered through this policy. In the material published some information may be duplicated or the same work may be reported from varied points of view. But this is a slight disadvantage, and one which cannot be obviated in reproduction from original sources. On the other hand, the expression of divergent opinions may prove of distinct value. The problems are new and a new science of dealing with the adult cripple is being brought into being. New authorities are springing up to meet the demands of national crisis, and the very stress of the situation is producing exceptional results. But the methods developed and the experience gained will be of lasting advantage to the cripple. The editor hopes to have appear in the columns of the journal during the coming year information requisite to an intelligent consideration of problems concerning the disabled soldier."

If it were necessary to comment on the excellence of such purposes, emphasis could be laid on the inclusion here of foreign material in English translation.

The Battle Gound for Wounded Men. In a rather colloquial article, Kellogg presents a most interesting survey of the agencies operative in Canada for the returned crippled. Beginning with the overseas return transportation there is a brief account of the civilian commission under which this is accomplished: "Almost by accident, Canada put into the hands of a civilian commission the handling of this return current of men from overseas. The far-reaching social significance of so doing is only now beginning to lay hold of the public. In testifying before a parliament committee in March the general charged with mustering battalions in Montreal district said tersely that he had no time to consider the handling of returned convalescents—his business was to produce fighting men for the front. Not only do the currents run in opposite directions, but their whole functioning is different. The goal of the Military Hospitals Commission is to take the discards of war and readjust them physically, vocationally, and spiritually to civil life. Gradually as the return current grows in volume, and as the commission becomes better known, its work is being visualized as a great economic and patriotic responsibility and service."

"The Canadian Army Medical Corps had been developed for fifteen years prior to the war. Base and camp hospitals were put in operation to care for the heavy medical work incidental to mustering the expeditionary force. Provision for physical examination of recruits had to be expanded, reorganized and brought to new efficiency following the

rejection of large numbers of unfit men after they had reached England. The corps was constantly drained of some of its most experienced physicians who accompanied the troops overseas. But even had the medical corps been equipped to handle invalided men also, so far as numbers go—and this is a moot point—it is my conviction that the present Canadian system which has vested administrative responsibility for handling returned men in a separate civilian agency is the better one. The physical restoration of a sick or temporarily injured soldier, so that he can return to the ranks, is a job for army doctor and drill master. The rehabilitation of a permanently injured man for the resumption of civilian life where he left off, or even the care of the wide variety of war wrecks who come back from the front, calls into play all manner of specialists, surgeons, psychiatrists, tuberculosis experts, trade teachers, agriculturists and the like."

The following subjects are then considered in some detail: war consumptives, hospitals for convalescents, vocational work . . . reëducation, "facing life again." The article makes no pretense at statistical or official statements, and yet by means of individual cases and "human" episodes brings to the fore a tangible picture which a more elaborate treatise might fail to do. There is thus carried an appeal to the social worker at large, the importance of which need not be mentioned.

The Reëducation and Placement of War Cripples. This is a ten-page account of largely personal observations concerning the work for crippled soldiers in France. Of all the Allies, this country earliest faced and met the necessity of constructive work on these lines, with the result that today familiarity with her experience and methods is almost fundamental to the best-directed efforts wherever undertaken.

Report of the Disabled Sailors' and Soldiers' Committee, Local Government Board of Great Britain. The most interesting feature of this report is the following table, giving figures under date of May 4, 1915:

	Army.	Navy.	Total.
Eyesight cases	245	9	254
Wounds and injuries to leg (necessitating amputation)	205	10	215
Wounds and injuries to arm (necessitating amputation)	170	6	176
Wounds and injuries to hand (necessitating amputation)	15	6	21
Wounds and injuries to leg (not necessitating amputation)	277	9	286
Wounds and injuries to arm (not necessitating amputation)	272	3	275
Wounds and injuries to hand (not necessitating amputation of complete hand)	224	11	235
Wounds and injuries to head	123	4	127
Herniæ	96	5	101
Miscellaneous wounds and injuries (not included in above)	129	6	135
Chest complaints (including 200 cases of tubercle of the lungs)	298	4	302
Rheumatism	116	6	122
Heart disease	284	—	284
Epilepsy	47	—	47
Nervous diseases	54	11	65
Insanity	29	—	29
Deafness	134	—	134
Frost-bite	6	—	6
Miscellaneous disabilities	150	13	163
Total	2874	103	2977

The Royal Orthopedic Reserve Hospital at Nürnberg, Germany. Under this heading appear three short descriptions of work of rehabilitation in Germany. Although published two years ago (1915) the paucity of data from such sources makes the text worth reading, though there is adduced nothing not already familiar to workers elsewhere. The articles are rather cursory, with emphasis on certain broadly social details, such as earnings and internal business arrangements. The article concludes with original recommendations, long since adopted in principle and amplified.

The Problem of the Disabled Soldier. This article sounds the surest note of any in the current number, and will repay perusal by anyone. With very elemental analysis it considers the precise conditions under which Canada put her forces in the field and proceeds to indicate with a kind of logical synthesis, the general constructive needs to be supplied.

In many respects the principles discussed have closer application to the United States than do analogous principles pertaining to France, England or other countries. Dealing in generalties only the article is one of the most fundamental expositions of the whole problem concerning care of the crippled which have recently come to light and will bear quoting at some length, particularly as some striking paragraphs are nearly complete in themselves and give food for thought.

"It is an interesting commentary upon the nature of the question and upon its importance that the warring nations have adopted measures that are almost identical. Differences in the measures adopted depend not upon any fundamental difference in the principles underlying them but upon differences in the social organization of the nation for which they are designed. Like many other nations, Canada provides an elaborate machinery for removing a man from civil to military life. She is like other nations, also, in that the machinery by which she returns the men of her forces to civilian life is less perfect than that by which they are recruited. The situation should be reversed. The man who joins an armed force is assured a position so long as he remains a soldier or sailor. The man who leaves an army or navy becomes a civilian. At once he is thrown upon his own resources and must commence to gain a livelihood in competition with his fellows; in fairness, no man should be subjected to such hazard until he is fitted to cope with it. This is like no preceding war. It is a war of nations, not of armies. As a result, old methods of providing for ex-soldiers have been found inadequate and new legislation to meet new conditions is being devised and enforced. France and England, for example, are remodelling their laws; Canada which had practically no laws for dealing with ex-soldiers when war commenced, will find it necessary to devise a whole procedure for that purpose. The laws by which France is providing for the return to civilian life of those who have served in her 'armies of the land and sea' are characteristically detailed in their provisions. They are governed in their design by principles similar to those which define the obligation to Canada toward her citizen soldiers and sailors. There is great similiarity between these principles and those underlying much of the recent legislation providing compensation for working men injured at their employment. There is sound reason in that similarity. Modern workmen's compensation

laws, such as those of the Province of Ontario, look upon the charge for insuring workmen against unavoidable accident as an item in the cost of producing articles manufactured: about 86 per cent. of the industrial accidents occurring in Germany in 1887 were unavoidable. It follows that the cost of insurance should be added to the price of the article manufactured, and that it should be borne by the consumer; the insurance is a part of the cost of the commodity which he purchases. The manner in which French public opinion on these matters was formed to sound lines is very striking. At first there was a universal tendency to assume that there is nothing left for a disabled man but a lifetime of stagnation as an idle pensioner. A definite policy of public instruction was commenced. In it every method of conveying information was used with all the prestige and authority that official approval could lend. Newspapers, magazines, posters, clergy, trades unions, manufacturers' associations, boards of trade, public service corporations, all united in insisting upon the dual obligation existing between the State and its citizens: there is an obligation upon the State to ensure an independent position to those who have been disabled in its service; and there is an obligation upon the citizen, both to be self-supporting in the measure of the ability remaining to him and to receive from his fellow-citizens no more than is his due. It is much less usual now for a disabled soldier to refuse the treatment by which his disability might be lessened or for him to decline the vocational training by which he might be made self-supporting. All Canadians, soldiers and others, should understand that disabled men who refuse to take advantage of the opportunities for treatment and for training offered to them do so at grave cost to themselves. A man who unreasonably refuses to accept the treatment and training made desirable by his disability penalizes himself; by his own choice he remains less capable of supporting himself than he might be. Moreover, such a man cannot expect to be pensioned for the entire disability existing in him; but only for that portion of the disability which would exist were he to accept reasonable treatment. In France, indeed, it is anticipated by many that the acceptance of appropriate treatment and of appropriate vocational training will be made a military necessity for disabled men. The inadequacy of our educational system becomes very apparent when a full-grown Canadian, a returned soldier, about to leave a hospital cured of a transient disablement, begs to be retained for a little longer in order that his primary education may be completed. If it is done for the disabled soldier, since the lack of education was in nowise dependent upon his military service, a means of obtaining knowledge should exist for every citizen who desires it. A civilian suffering from tuberculosis is permitted, restrained by nothing but the degree of his incapacity, to circulate among his fellows, often to his own detriment and to the danger of those among whom he moves. Can advantage be taken of the peculiar situation of soldiers or sailors suffering from tuberculosis to place restrictions upon them—for the benefit of themselves and their fellows—which are not placed upon civilians? It should be done for soldiers; it is but one of the things which should be done for us all under the authority of a not-yet-established Federal Department of Public Health. If these and similar hardships, unconnected with military service, affecting disabled men, be dealt with

adequately, the first step will have been taken toward remedying some of those defects in our national organization which stress of war has made very plain."

War Cripples in Austria and Germany. This is a superficial but interesting survey of the efforts of the chief Central Powers toward rehabilitation of the crippled. It emphasizes, among other things, the enormous losses in the Central Army and the fact that in Austria as well as in Germany the authorities do their utmost to keep cripples out of sight in populated centers. It mentions the "Cripples' Town" outside of Vienna where, in November, 1915, there were nearly 4000 maimed soldiers in more or less permanent residence.

"Supposing an unfortunate man, both of whose legs had been amputated, to arrive in 'Cripples' Town,' it is found by experience that, by the fact of his being with so many others, all of whom are afflicted with the loss of one or more limbs, he is encouraged to try to make the best of his infirmities. In some cases maimed men are actually employed to teach others, showing them what it is possible for a cripple to do, with determination, courage and taking pains. A man with both legs amputated, for instance, is given stumps, and then with the help of a moving rope fastened to a roller he gradually learns to walk again along the hospital passages. When he has learned to walk quite quickly on the low artificial legs he is given higher ones, and so higher and higher until he reaches his natural height once more. The next stage in his training is to learn to use artificial legs with movable knee-joints, so that he can sit and kneel down. Finally, he receives artificial legs, with not only movable knee-joints but also ankle-joints provided with strong springs, so that his gait acquires elasticity. Thus equipped he practises walking about in the hospital garden with no other help than that of a light cane. It is not enough, however, for these crippled soldiers to learn to walk again, or even to dance. They must learn to become once more useful members of society. Consequently, they enter one of the thirty-two workshops in 'Cripples' Town,' where they are put to do the work for which they are best fitted. A man who was a carpenter, for example, but who has a stiff elbow, is set to do planing as the speediest way of recovering the elasticity of the elbow. A locksmith is put to use a file; a man with a stiff shoulder must try to use a saw, and a stiff-fingered man to weave baskets. In Germany and Austria, however, this is becoming a veritable art, and the artificial limbs of before the war are now wholly out of date, to such pitch of perfection have those of today been brought." R. P.

Bone-graft Surgery: Its Application to Fracture Caused by Modern Projectiles.—DOWNER (*Military Surgeon*, September, 1917) gives clinical experiences from Serbia and Russia. The most perplexing problems encountered by the military surgeon are the compound comminuted fractures when there is loss of bone substance. These necessitate the transplantation of living bone to act as an internal fixation splint and to stimulate the formation of new bone. In modelling the bone-graft into wedges and pins and inlays, and in fashioning groove joints and dovetail joints the surgeon needs to have had, in advance, good mechani-

cal training in working with tools. The electric current is available nearly everywhere, not only in towns and cities, but also with the armies. This makes it possible to use motor-driven bone surgery outfits which increase the skill and accuracy of the surgeon. A good motor outfit and a strong extension apparatus are indispensable to the bone surgeon at the front. At the beginning of the war amputations were performed almost indiscriminately upon all the fronts. Later, during the second year of the war, more conservative treatment was used, and there was a great decrease in the number of amputations. Many cases, however, were treated too conservatively. Thus badly shattered legs were placed at once in plaster casts, no attempt being made at drainage, and the wounds of entrance and exit being left as they were found upon the battlefield. Often such casts were left on for six weeks, and when removed great destruction of tissues was found and the casts were full of pus. The writer finds that the conservative treatment is still being overdone. The result of these methods he sees in many of the cases which he has operated on. There had been no incision, no drainage, except that made by the projectile, and the parts had been immobilized by some external fixation appliance. The wounds had been allowed to suppurate, the wounds of entrance and exit becoming mere pin-point openings, through which a small amount of pus was steadily oozing. Upon opening such wounds there were found many fragments of dead and diseased bone, surrounded by much dense fibrous tissue, resulting from the chronic inflammation. Not only were the fragments dead, but also the fractured ends of the bones had begun to soften, and that there was very little callus formation. Very few cases of comminuted compound fractures treated in this way turn out successfully. The writer thinks it preferable to remove all sequestra at the start, leaving a free drainage, then the application of a plaster cast, with a window cut in, and the wound dressed daily through the window. When all suppuration has ceased a bone-graft should be taken, usually from the tibia, and inserted by the inlay method. With the electrically-driven outfit a suitable graft can be secured in ten to fifteen minutes, and then fashioned very quickly to fit the place for which it is intended. The writer has seen evidence for both the views which are advanced as to the role of the graft. In some cases it has acted merely as a scaffold, all the new bone coming from the host. In one case, however, in which the graft was fractured the fractured ends reunited without the aid of outside tissue, so that in this example the graft displayed osteogenetic powers. At any rate it may safely be said that the graft acts as a stimulant, and even when placed in infected areas it causes the formation of new healthy bone. It is much better than a metal plate in many cases, for this has been found to inhibit the formation of bone callus. In performing the operation it is well to use two separate groups of instruments, one for making ready the diseased area and the other for preparing the graft. The plaster casts are left on for four or five months, being changed about once a month. After the removal of the cast, massage and usual post-fracture treatment is begun. The results are so good that so long as there is any healthy host bone left in a limb one is justified in giving bone transplantation a trial. W. H. F. A.

Triple Typhoid Vaccine (Bacillus Typhosus, B. Paratyphosus A, and B. Paratyphosus B).—CRAIG (*Jour. Am. Med. Assn.*, September 22, 1917, lxix, No. 12, p. 1000) states that the necessity for protecting our overseas troops against the paratyphoid fevers has rendered inoculations against B. typhosus A and B. typhosus B imperative, and the fact that these infections occur also in our own country, and will undoubtedly appear sooner or later in our large cantonments, made it advisable that the entire army should be inoculated as a prophylactic measure. A triple vaccine is prepared at the Army Medical School, Washington, D. C., which contains 1,000,000,000 typhoid bacilli, 750,000,000 paratyphoid A bacilli, and 750,000,000 paratyphoid B bacilli per cubic centimeter. The first dose is 0.5 c.c. and the second and third each 1 c.c. The inoculations are made subcutaneously and at intervals of seven days. Craig concludes, as a result of his studies, that the triple typhoid vaccine, which is the vaccine now adopted for immunizing our entire army against typhoid and paratyphoid fevers, gives as good results in immunization, so far as can be judged by agglutinin curves, as does the typhoid vaccine alone, and that neither the general nor the local reactions following its injection are different in any degrees from those occurring after the inoculation of simple typhoid vaccine. The results, both from a practical and from a theoretical point of view, are all in favor of combining the three organisms in a single vaccine, and it is believed amply justify the use of the triple typhoid vaccine in immunizing our troops against typhoid and paratyphoid fevers. J. M. McC.

The Relation of Psychology to Military Activities.—YERKES (*Mental Hygiene*, July, 1917) suggests briefly a few of the varied lines of service in which the psychologists may be of value in the conduct of the war. In recruiting they have an important special task, namely, of classifying men according to their mental characteristics and of indicating their degree of adequacy for military training or special work in the military organization. Current methods of mental examining are unsuited to military demands, and consequently new and specially adapted methods must be prepared if the results are to be trustworthy and practically serviceable. In examining recruits it is their prospective function to help eliminate those who cannot render service worthy of their hire; to ascertain various degrees and kinds of special ability so that the individual shall be placed in a position of maximum usefulness; and to detect those who by reason of mental instability or psychopathic condition demand the attention of the medical expert. Already experimental psychological studies, by means of appropriate measurements, are being made on men being trained in naval gunnery and on men receiving preparatory training in aviation. By means of such studies it is believed that information will be derived which will be of value in selecting men suitable for these services and in discovering individuals who will improve most satisfactorily under training. W. H. F. A.

REVIEWS

THE MEDICAL CLINICS OF NORTH AMERICA. Philadelphia Number, Vol. I, No. 2. Pp. 195 to 464. Philadelphia and London: W. B. Saunders Company, 1917.

THIS number presents clinics from the University of Pennsylvania, the Jefferson, the Pennsylvania, the Philadelphia General and the Polyclinic Hospitals of Philadelphia. Among its contributors are twelve internists, a roentgenologist, a serologist, a neurologist and a dermatologist. Some of their articles consist of true bedside clinics, such as are daily given to medical students. Others are more formal presentations of cases, with extensive reference to the literature, and still others are not clinics at all but contributions of a more general nature without any reference to individual patients. The latter are represented by Dr. T. H. Weisenburg's article on poliomyelitis and Dr. John A. Kolmer's article on the diagnostic value of examinations of the cerebrospinal fluid.

Dr. Thomas McCrae, in the opening chapter, analyzes 3 cases of aortitis, placing emphasis upon the rheumatic as well as the luetic etiology. In Dr. Alfred Stengel's contribution is given a workable classification of the nephritides based upon the newer knowledge of these conditions, but emphasizing the clinical rather than the laboratory diagnostic features. Dr. Hobart Amory Hare discusses the indications, and particularly the contra-indications, to the use of digitalis in heart disease. Aortic aneurysm and myelogenous leukemia are illustrated by patients from the clinic of Dr. Joseph Sailer. Dr. David Riesman makes use of the unusual opportunities of the Philadelphia General Hospital in presenting a case of progressive myocarditis. Dr. Henry K. Pancoast discusses in his usual conservative manner the value of the roentgen rays as a diagnostic aid in pulmonary tuberculosis and calls attention to the similarity of the fibrosis of this disease to that produced by excessive dust inhalation. Angina pectoris and its allied conditions are presented by Dr. A. A. Stevens. A somewhat exhaustive clinic upon the cardiac arrhythmias is given by Dr. Ross V. Patterson, while Dr. John H. Musser, Jr., describes an interesting case of auricular fibrillation of long duration. Gastric infection and ulcerative endocarditis are treated by Dr. Martin Rehfuss and Dr. Judson Daland respectively. Dr. O. H. Perry Pepper discusses the essentials and limitations of an

average diet. A chapter by Dr. Jay Frank Schamberg upon the causes of reaction after salvarsan should be read by all who use it or allied preparations. Dr. Elmer H. Funk concludes the number with an interesting case of osteitis deformans. T. G. M.

ANNUAL REPORT OF THE SURGEON-GENERAL OF THE PUBLIC HEALTH SERVICE OF THE UNITED STATES FOR THE FISCAL YEAR, 1916.

THESE reports speak for themselves always and necessarily do not permit of detailed description. In this report the pellagra and plague surveys are particularly interesting, however. The problem of vocational diseases and industrial hygiene are considered, but only a beginning has been made in this work. It is interesting to note in the report of studies of child labor problems in Massachusetts in relation to health that diseases and defects of a more serious character are less common among minor cotton-mill employees than in the general school population of the same ages.

The present volume contains reports on (1) scientific research; (2) foreign and insular quarantine and immigration (maritime quarantine); (3) domestic (interstate) quarantine; (4) sanitary reports and statistics; (5) marine hospitals and relief; (6) personnel and accounts; (7) miscellaneous. C. W. S.

THE PRINCIPLES OF ACIDOSIS AND CLINICAL METHODS FOR ITS STUDY. By ANDREW WATSON SELLARDS, Associate in Harvard Medical School Department of Tropical Medicine. Pp. 117. Cambridge-Harvard University Press, 1917.

THIS book is essentially an analysis of the more important and representative articles on acidosis, and is a most excellent presentation of the subject. Clear and succinct in detail, and with a positiveness in deduction, it is very readable and leads through the bewildering choas of the literature in a no uncertain manner so far as our present knowledge permits. The places where our information is still incomplete are pointed out with equal detail.

Following a short historical note he reviews the equilibrium between acids and bases and the methods of diagnosing acidosis. The determination of the tolerance to sodium bicarbonate he maintains is the most delicate test we have of acidosis, but presents fully the relative value of the various tests with their respective technics.

Regarding the definition of acidosis, he deplores the confusing subdivisions and broadly defines the term as a depletion of the

blood and other tissues of the body in fixed bases. Three conditions are known in which severe acidosis occurs, namely, diabetes, some of the nephropathies and the so-called "food intoxication" of children, and these he described fully. He refers but little to the role acidosis may play in the untoward symptoms following general anesthesia. However, his opportunities for studying acidosis in Asiatic cholera while in Manila permit interesting observations which open up new problems.

The therapy of acidosis and the technic of sterilizing solutions of sodium bicarbonate are described fully. C. W. S.

ELEMENTS OF HYGIENE AND PUBLIC HEALTH. A TEXT-BOOK FOR STUDENTS AND PRACTITIONERS OF MEDICINE. By CHARLES PORTER, Medical Officer of Health, London. Pp. 384. New York: Oxford University Press, 1917.

WRITTEN in a most interesting style common to so many of the English writers, this book will be welcomed not only by physicians, but also by all others who are interested in public health. All through the book special emphasis is laid upon the role the practising physician should assume in community health problems. Too little stress has been made of this point heretofore, the effort having been made more to organize official medical bureaus. As an incentive to secure the coöperation of family physicians, mention is made of the custom in London of paying small fees by the city for the report of communicable diseases.

The first chapter of the book deals with personal hygiene, and is an excellent presentation of the subject, with the possible criticism of his advice to sweeten the water given to infants between feedings, a practice not approved by pediatricians in this country. Then follow several chapters on public health and disease, tropical diseases and animal parasites. A chapter on occupational diseases is of particular interest to physicians of this country at this time, for we are beginning to concern ourselves more and more with this problem. The section on food is excellent. It concerns itself principally with the detection of food poisonous to man and the nature of such poisoning. The chapter on climate and meteorology, however, is elementary, and it is difficult to see the necessity of incorporating such detail in a work of this kind. The air, the soil, water, housing and ventilation, the disposal of refuse and waste are adequately considered in separate chapters.

The book ends with a clear review of the method of computing vital statistics and pitfalls to avoid in interpreting such statistics. An appendix includes copies of leaflets and cards for distribution concerning the care of the baby and contagious diseases.

C. W. S.

PAPERS ON THE INFLUENCE OF SMOKE ON HEALTH. Edited by OSKAR KLOTZ, M.D., and WILLIAM CHARLES WHITE, M.D., Mellon Institute of Industrial Research and School of Specific Industries. Bulletin No. 9, University of Pittsburgh.

THIS bulletin contains the work done by the physicians and laboratory investigators on the staff of the smoke investigation committee of the Mellon Institute of Industrial Research.

Dr. Cohoe's paper on the "Relation of Atmospheric Smoke and Health" gives a review of the literature on the subject, with the vital statistics of communities noted for their smoke. He wisely does not attempt any definite conclusion from a statistical study, but shows unmistakably the marked influence of the smoke in raising the death-rate in acute lung diseases.

Dr. Oskar Klotz's paper on "Pulmonary Anthracosis" is a complete pathological study. The work of Doctors Holman and Haythorn are valuable contributions containing results of original research. Dr. Holman, studying the bacteriology of soot, concludes that soot has some germicidal action, but also as it occurs in smoke, clouds, fogs, etc., protects microörganisms from the destructive action of the sunlight. Dr. Haythorn's study of histological evidences is worthy of particular attention. He concludes that in tuberculosis an anthracotic condition may actively assist healing, in that it is an additional stimulus to fibrosis; but in acute inflammatory conditions an anthracotic condition becomes seriously detrimental because of the obliteration of the lymphatic spaces.

The papers make up an exhaustive study, and any future investigation must take cognizance of this work. C. W. S.

INDEX OF DIFFERENTIAL DIAGNOSIS. By HERBERT FRENCH, M.A., M.D. (Oxon.), F.R.C.P. (Lond.). Second edition. Pp. 911; 37 colored plates and over 300 illustrations in the text. New York: William Wood & Company, 1917.

THE purpose of this book is clearly stated in its title, and the work must be estimated and used in accordance with its expressed purpose. It is an index of differential diagnosis and nothing more. To the question as to whether there is an excuse or need for such an index the repeated printings of the first edition and now the appearance of an enlarged second edition would seem to give sufficient answer.

It is a large book and profusely illustrated with well-chosen and extremely well-reproduced plates, drawings and charts. The many photographs are especially clear and the reproduction of colored photographs are admirable. Apparently nothing is omitted, as

might be suggested by a closely printed index covering almost forty pages, with four columns to the page.

In a month's use as a reference book no serious errors have been found, and except for the very frequent error of putting an umlaut over Prof. Duhring's name on page 219 and a misprint in the page number in the indexing of Trichina, no typographical errors were observed. O. H. P. P.

OBSTETRICS FOR NURSES. By CHARLES B. REED, M.D., Obstetrician to Wesley Memorial Hospital, Chicago. Pp. 374; 130 illustrations. St. Louis: C. V. Mosby Company, 1917.

THIS book is a practical manual of obstetrics for nurses. The subject is taken up in the following order: A small amount of space is given to anatomy and physiology, followed by normal pregnancy, abnormal pregnancy, labor, the care of the patient during labor and puerperium, operations, complications, infection, the care of the child, infant feeding, a list of diets, solutions and therapeutic index, and a glossary. The book contains good illustrations. The fetal circulation, which many authors fail to give, is described so clearly that any nurse may understand. Dr. Reed endorses the use of nitrous oxide gas in the first part of the second stage of labor, but advises ether or chloroform for the completion of the second stage. The section on the care of the child does not compare favorably with the rest of the book. The care of the premature infant might have been described more fully. The book will be found of practical value for those for whom it is intended. S. B. S.

DISEASES OF THE SKIN. By RICHARD T. SUTTON, M.D., Professor of Diseases of the Skin, University of Kansas, School of Medicine, etc. Second edition. Pp. 1021; 833 illustrations. St. Louis: C. V. Mosby Company, 1917.

THIS volume is a large, well-made book. The reviewer not having the privilege of reading the first edition has no means of comparing it with the later edition, but must rely entirely upon his perusal of the volume at hand for his comment. Unfortunately, by reason of his close personal contact with the work and works of Duhring, Stelwagon, Hartzell, Van Harlingen, and Schamberg, representing the so-called Philadelphia school of dermatology, he is too likely to criticize this book unduly severely. In a word, while it is large in size and most formidable in appearance, it may be said not to measure up well to these standard works, although it seems to have been the aim of the author to do so. Pictures, and this term is used advisedly, should make up a large part of any book on dermatology,

but they should be good ones. Here we find the conventional anatomical diagrams, as usual, very good—the photomicrographs or the microphotographs, as the case may be, rather vague in their demonstrating features—and the colored plates of rather indifferent value. The pictures intended to illustrate diseases are very profuse in number but poor in quality, and thereby lose their instructive value. There is also a great abundance of references, but where an author is referred to, there is no means of knowing just what point in the discussion of the disease is attributed to that particular author. Using authorities in that manner renders the value of the bibliography questionable. Here and there throughout the sections on treatment we note obsolete forms of prescription writing. Any formula which has petrolatum for a base, for instance, does not require lengthy Latin or near-Latin instructions to make an ointment out of the ingredients named. It looks imposing but it is unnecessary. The introductory material is especially well handled, that is to say, the anatomy and physiology of the skin and the general etiology and pathology of skin diseases. Usually these chapters in works of this character are awful "bores" and contain all the fables and traditions from Hali Abbas down. Sutton is to be congratulated on his frank denial of "diathesis" and his acceptance and elaboration of occupation as a factor of prime importance in etiology. The relation of proteid substances, toxins and antitoxins to the causation of skin diseases is ably handled. Grouping of the primary lesions as to regional distribution for diagnostic purposes seems unnecessary, however, even if not inaccurate. To our mind and eye, any of the primary lesions not only may but do occur everywhere where the skin is. While this section is rather elaborate, opportunity was neglected to observe the diagnostic significance of pustules in the palms of the hand, on the female breast and on the shaft of the penis in scabies, and the pronounced distribution of the lesions in the different forms of pediculosis. Under general consideration of treatment, Sutton remarks that "purely local cutaneous maladies are indeed few and far between," with considerable emphasis, as though this were the most modern thought in this field. He qualifies this later by the statement that "constitutional treatment is intended more for the purpose of increasing the bodily resistance of the patient than for any direct or specific effect it may have on the causative factor." The relation between the general health and skin diseases is always a troublesome topic to handle, since just as we have the matter conclusively proved something embarrassing happens. We refer in passing to the everlasting enigma "psoriasis," to awaken reminiscences in the reader's mind. Looking over the book, as a whole, it may be said to include all the legends and mythology of dermatology with the modern frills of treatment, and countless illustrations of indifferent value appended. S. H. B.

PROGRESS
OF
MEDICAL SCIENCE

SURGERY

UNDER THE CHARGE OF

T. TURNER THOMAS, M.D.,

ASSOCIATE PROFESSOR OF APPLIED ANATOMY AND ASSOCIATE IN SURGERY IN THE UNIVERSITY OF PENNSYLVANIA; SURGEON TO THE PHILADELPHIA GENERAL HOSPITAL AND ASSISTANT SURGEON TO THE UNIVERSITY HOSPITAL.

Wound Treatment.—DONALDSON and JOYCE (*Lancet*, ii, 1917, 445) say that three main methods of wound treatment are in vogue, the antiseptic, the physiological and the surgical, namely, by excision of the wound. The last named has come into vogue because of the disappointing results obtained from the first two, on the score of their inefficiency or because of the time element. From the physiological or hypertonic saline method has evolved as an outshoot the salt-pack treatment of wounds, for which its advocates claim extraordinarily good results. These include the simplicity of method, the avoidance of daily dressings of the patient, the rapidity with which the wound becomes a healthy granulating surface, the absence of secondary hemorrhage, and the rapid general improvement of the patient. All this means a considerable curtailment of the time spent by the wounded man in hospital. Hitherto the good results have been attributed in some obscure way to the presence of the salt acting more or less on the physiological line described by Wright, but it is felt by all who have written about salt-packs that some other explanation is necessary. Donaldson and Joyce afford what they believe to be the true explanation of the phenomena observed, and believe that it will clear up many of the points that have hitherto been obscure. The key to the situation depends on two observations—one clinical and the other bacteriological. The former consists in the fact that cases which smell (and the odor is one of the most characteristic features of these cases) do well, while those which do not make no headway. The bacteriological observation consists in the discovery that a certain bacillus is apparently constant in the wounds which emit the odor, while it is absent or cannot be recovered from those which do not smell. The bacillus is a spore-bearing anaërobe of a saprophytic nature and belongs to the proteolytic group of organisms, the group which includes B. tetani and B. edematous maligni. Unlike these

latter it appears to be non-pathogenic to the animals experimented on, and, what is of more importance, appears to be non-pathogenic for man when introduced into wounds. Further, unlike the above-named pathogenic varieties it does not appear to set free any toxins injurious to the patient in the course of its action on the dead tissues. It acts, apparently, in virtue of its proteolytic powers, only on devitalized tissues, and possibly on toxalbumins, and appears to possess no power of attacking healthy structures. The surgical or excision treatment of wounds aims at removing only microscopically devitalized tissues, and in so doing inflicts a fresh trauma liable to reinfection. It is proposed here to substitute this, the Reading bacillus in a living form, and it is probably present in most of the wounds if given a chance to carry out its functions. These are directed toward the removal not only of the grossly damaged tissue, but it succeeds also in attacking the microscopically damaged structures. As a result the body forces are freed from the constant menace of septic poisoning, thus allowing them to begin the process of repair. Examples are given of gunshot wounds cases which have been treated in various ways previously, but without success. These, however, on being sowed later with living cultures of the bacillus have immediately started to do well and have ended in rapid recovery. The organism is probably present in the large number of infected wounds, but requires more or less anaërobic conditions before it can come into play. It is essential that the wound be thoroughly well laid open in the first instance, exposing every pocket and sinus, so that the packing may completely fill the wound.

The Handling of Early and Doubtful Cases of Cancer.—GREENOUGH (*Ann. Surg.*, 1917, lxvi, 385), basing his conclusions upon the answers to a circular letter to a large number of representative members of the profession, says that the opposition to the use of the exploratory incision for the removal of tissue suspected of malignancy is very real. Many surgeons believe it is never necessary or advisable; others, and they are the majority, believe that in certain situations it is permissible when all other resources of diagnosis (in which should be counted the benefits of consultation) have been exhausted, and even then only (1) when the tumor is a superficial one, or (2) when a frozen section can be obtained immediately and the operation completed under one anesthesia. Under these conditions, and under these conditions only, can the exploratory incision of the tumor tissue be justified as a reasonable surgical procedure. If exploratory removal of tissue is to be discountenanced, what becomes of the project of the free diagnosis of suspected cancer tissue? Greenough believes it assumes an importance greater than ever, because it should be applied to all pathological tissues removed for the cure of disease. The function should be a State function, and the report should go to the patient or his friends as well as to the surgeon. It should be the final means of checking up the end-results of the surgical diagnosis and treatment of malignant disease. Only by some such drastic measures as this can the incompetent surgeon be prevented from depriving the patient of his only hope of cure of cancer by an early operation. In general, superficial and ulcerated lesions are the only ones in which fragments of tissue

may be safely removed for microscopic diagnosis. In deep tumors, where the exploratory incision necessarily opens up normal tissue to infection, exploratory excision of suspected tissue is to be condemned, and should be avoided. Where other resources of diagnosis have been exhausted, and an exploratory incision, with an immediate frozen section diagnosis, and immediate performance of the radical operation if it prove necessary, is the least dangerous procedure for the patient. No suspected tissue should be excised for diagnosis, unless by a surgeon who is equipped to perform immediately the radical operation for the cure of cancer of the organ involved. The routine pathological examination of all tumor tissues removed by operation should be made compulsory. To this end competent laboratories for the free diagnosis of pathological material should be maintained as a function of the State.

Posture in Cases of Abdominal Drainage. — HILL (*Ann. Surg.*, 1917, lxvi, 414) says that abdominal drainage with the patient in the prone position has been tried out at the St. Louis City Hospital so successfully that a number of the staff now use it as a matter of routine. As it is now carried out the patient is placed on the abdomen usually for from twenty-four to forty-eight hours, with the head of the bed elevated about ten to twelve inches. A pillow is placed under the lower part of the chest and one is placed under the head, so as to give the patient ample room to breathe. The principal objection to this position is that it is not comfortable. Their observations lead them to believe, however, that it is not nearly as uncomfortable as one would suppose. By this position are obtained the maximum effects of gravity, intra-abdominal pressure, and capillary attraction. In addition to this there are no spaces in the front of the abdomen to favor the formation of pockets, as there are in the pelvis and alongside of the spine. The pus is also brought against a part of the abdomen where bloodvessels and lymphatics are not nearly so numerous as they are in the pelvis. The lateral position, in which the patient has been placed on the right side, has been found to be very efficient. The head of the bed is slightly elevated, and a pillow is placed under the region of the liver, so as to prevent any space for accumulation of fluid in the kidney region. The patient is placed far enough over so that pus will drain from in front of the left kidney. In an analysis of 104 drainage cases operated on in this hospital during the last year and a half the three positions have been employed with sufficient frequency to warrant drawing some conclusion as to the relative merits of each. In a series of 15 cases of appendicitis of various grades of severity treated in the lateral position there was an absence of mortality. In another series of 42 cases of appendicitis treated in the abdominal position there were but two deaths. In the 47 cases of the same condition treated in the Fowler position there were five deaths.

Ultimate Results Following Nephropexy in Cases of Symptomatic Nephroptosis.—CLARK and BLOCK (*Ann. Surg.*, lxvi, 479) present a study of 50 cases of nephroptosis operated on since 1900. They show a steady diminution in the percentage of elective cases. If one adheres to the rule that surgical intervention is indicated only when the kidney or the bladder gives expression to symptoms the ratio of cure

will compare favorably with that following other approved operative measures. They have settled upon three cardinal indications for possible surgical intervention in movable or floating kidneys. These are fixed pain in the renal area, Dietl's crisis, and marked vesical distress referred from the renal area. Even these symptoms, however clear, must be supplemented and confirmed by a rigid clinical investigation, which should include a cystoscopic examination, catheterization of the ureter on the affected side, and, in the majority of cases, an injection of thorium and a roentgen-ray examination to ascertain the degree of dilatation of the renal pelvis. An appended table of cases operated upon since 1910, the period during which they have endeavored to select only those of a true pathological nephroptosis, with a summary of symptoms, with diagnosis, operation, and ultimate results, present a 70 per cent. restoration to active efficiency of the women operated on, a degree of restoration which compares favorably with many other operations generally accepted by representative surgeons. The kidney, particularly the right, is not a fixed organ, and even an excessive range of mobility is no certain index of a reflex gastric or nervous disability unless direct renal symptoms referable to the kidney are present. No diagnosis should rest solely upon palpation of an unduly movable kidney or upon a definite symptomatology unless this is confirmed by a cystoscopic examination, catheterization of the ureters, and a pyelograph to define the point of kinking in the ureter and the degree of dilatation of the renal pelvis. In their hands the Edebohls method, with a slight modification, has proved satisfactory as a permanent means of anchorage.

Influence of Sun's Rays on Bone and Joint Tuberculosis.—FREIBERG (*Am. Jour. Orthrop. Surg.*, 1917, xv, 625) says from a study of 4 cases that from his experience with these 3 cases it seems inevitable to conclude that exposure to the sun's rays as here practised resulted in speedy improvement which can fairly be attributed to this agency. The retrogression which ensued upon having to abandon the systemic use of the sunlight seems to emphasize this fact and makes it desirable to continue this treatment during the winter in his own environment. The results obtained seem to indicate the value of this method even in the vicinity of large cities and at low altitudes, where, according to Rollier, who originated this treatment, the potency of the ultraviolet rays is greatly diminished by the stratum of moist and unclean air through which they must pass. It is by no means certain that the ultraviolet rays are the essentially active or the only active part of the sunlight in a therapeutic sense. In Freiberg's experience the use of the quartz ultraviolet lamp, the so-called Alpine lamp, has failed to show that it may be considered even a fair substitute for sunlight. Its light, on the other hand, is quite rich in ultraviolet rays. It seems worth while to strive to construct a form of shelter for patients which will protect them from unendurable cold, and especially humid winds, thus making possible the continuation of treatment during bright days of the whole winter season. It seems necessary to expose the whole body in order to obtain the best results. Such shelters should therefore have as covers some material permitting the easy passage of the ultraviolet rays. An investigation in this direction is under way, but has not yet yielded any result of value.

THERAPEUTICS

UNDER THE CHARGE OF

SAMUEL W. LAMBERT, M.D.,

PROFESSOR OF CLINICAL MEDICINE IN THE COLLEGE OF PHYSICIANS AND SURGEONS, COLUMBIA UNIVERSITY, NEW YORK.

Optochin in Pneumonia.—STÜHMER (*Med. Klin.*, 1916, xii, 1286), as a result of his observations, believes that the administration of optochin is of value for the treatment of pneumonia in a certain percentage of cases. He found that in about 60 per cent. of cases it exerted a favorable influence and failed in about 40 per cent. The cases that failed to benefit by optochin therapy belonged to the more serious type of pneumonia, those which from the beginning were of doubtful outcome. He also observed that the administration of optochin is without value if a permanent fall in temperature amounting to at least 1° or 2° C. does not occur during the second day of administration. Stühmer prefers to administer the remedy by mouth in the form of the more soluble hydrochloride. He recommends as proper dosage 0.2 gram given at four-hour intervals throughout the twenty-four hours, a total of six doses daily. The patient should be watched carefully for tinnitus or impairment of hearing and the drug immediately discontinued upon the appearance of these early toxic symptoms. He observed one instance of total amaurosis following a total of 2.25 grams in a period of forty-eight hours. Patients that react favorably to optochin treatment show a fall of temperature that begins by lysis on the second day of the treatment and usually reaches normal by the fifth or sixth day. A marked beneficial effect upon the general symptoms, such as prostration, delirium and dyspnea, also followed the treatment. He enters into a theoretical explanation of the method of its beneficial action by attributing the improvement either to a simple antipyretic action or to a specific property of the drug to limit the action of the pneumococcus toxins in their ability to produce fever and general toxic symptoms. He also explains the failures of optochin therapy in certain cases by the theories that there are optochin-fast strains of pneumococci or that the drug is used in insufficient quantities and the consequent development of an optochin-fast strain results. Furthermore in some cases the drug may not reach the pneumococci in the lungs. The author has observed that empyema, extension of consolidation and relapses occur in patients under optochin treatment. He concludes that the drug is of some value for its antipyretic and symptomatic effects in pneumonia, but that it scarcely can be regarded as capable of improving the prognosis to life.

A Study of Exophthalmic Goitre from the Point of View of the Basal Metabolism.—MEANS and AUB (*Jour. Am. Med. Assn.*, 1917, lxix, 33) report the results of 224 observations on the basal metabolism in 55 cases of true toxic goitre and 20 observations in 18 cases of non-toxic goitre. They also made 37 observations on border-line cases and about

35 single observations in control cases, normal and pathological. The authors find that the toxicity as judged clinically runs very nearly parallel with the rise of metabolism. In the clinically non-toxic cases metabolism was practically normal; in the mildly toxic there was an average rise in basal metabolism of 43 per cent., in the moderately toxic of 53 per cent., and in the very toxic an average rise of 76 per cent. They concluded that the rise in basal metabolism furnishes an accurate index of the degree of toxicity in hyperthyroidism, and that it amounts to a functional test of the thyroid gland. They use their observations on basal metabolism as criteria for judging the severity and course of thyrotoxicosis and the effect of various methods of treatment. The chief therapeutic agents studied by Means and Aub were complete rest in bed, surgery, the roentgen rays, and the effect of a few drugs. It was found that rest in bed usually caused a marked fall in the basal metabolism (from +81 to +67), which continued over a period of from one to three weeks when a level or plateau was reached. This decrease in basal metabolism was not accentuated by the administration of quinin hydrobromide, veronal, paraldehyde, or triple bromides. The effect of the roentgen rays was variable: 6 patients received no treatment except the roentgen rays, and continued at their normal occupations; 2 showed no improvement; 2 were markedly benefited, and 2 showed slight improvement; 10 patients had complete rest in bed in addition to the irradiation; 6 of these showed either no improvement or else no more improvement than might be explained on the basis of rest; 2 others showed a moderate improvement. The roentgen ray proved most effective in reducing basal metabolism in the mild cases, least effective in the very severe cases. The effect of surgery was studied in 20 cases. In 12 cases observed just before and shortly after partial thyroidectomy, a striking fall in basal metabolism and toxicity occurred in every one. Some of the patients had had complete rest in bed and had reached the plateau before operation. In 6 of these the basal metabolism subsequently rose again. The effect of ligation of the thyroid arteries (2 cases) seemed to be a decrease in basal metabolism. The results of these observations suggest the following treatment of exophthalmic goitre: (*a*) Complete rest in bed plus irradiation should be continued until metabolism reaches a level. (*b*) If rest and the roentgen rays fail to restore metabolism to within 20 per cent. of the normal it is proper to resort to surgery unless there is some definite contra-indication; among contra-indications a rising metabolism, despite complete rest, seems to be very important. (*c*) Following operation, if metabolism again increases further active treatment should be carried out. Patients should be kept under observation for a long time—years rather than months.

A Method of Treatment of Mercuric Chloride Poisoning.—Weiss (*Jour. Am. Med. Assn.*, 1917, lxviii, 1618) reports a series of 28 cases with only one fatality. The treatment is based on the Fischer theory of nephritis. In reality the treatment is a modification of that recently offered by Lambert and Patterson. Weiss gives alkali, hypertonic salts by mouth, rectum, and intravenously. By mouth he uses "Imperial Drink," made up as follows: potassium bicarbonate, 60 grains; sodium citrate, 30 grains; sugar, 60 grains; lemon or orange juice to taste to 8 ounces of water. The salts are added just before the

drink is used. Intravenously, Fischer's solution is injected. The treatment begins when the patient arrives at the hospital, which is usually within a few hours after the mercury is taken. The stomach is washed with 1 quart of milk and the whites of 3 eggs, followed by water. Before the tube is removed 3 ounces of magnesium sulphate dissolved in 6 ounces of water is introduced into the stomach. A soap-suds enema is then given. The enema and the saline cathartic open the gastro-intestinal tract and so aid in the elimination of the mercury. If the patient failed to vomit immediately after taking the poison, or if he did not receive medical attention within three hours of swallowing the mercury, an intravenous injection of Fischer's solution is given at once; 1500 c.c. may be injected if there be no cardiac involvement. The patient receives 6 to 8 glasses of "Imperial Drink" a day and "is given large quantities of water by mouth." He is allowed a liberal diet—almost anything except an excess of protein food. The analysis of the urine is used as the control of the treatment. Large quantities of urine are voided because of the large fluid intake. The urine should be kept alkaline to methyl red, for Fischer has shown that if the urine of a nephritic patient cannot be made alkaline to methyl red the patient's condition is dangerous. The almost universal albuminuria clears up more or less rapidly when sufficient alkali is introduced into the body to counteract the abnormal amount of acids produced. If the amount of urine voided is close to normal and becomes alkaline on the medication by mouth within the first twelve to twenty-four hours the intravenous injection of Fischer's solution may be omitted. If the patient's urine continues acid to methyl red, intravenous injection must be given without delay and repeated if necessary. Usually the urine becomes alkaline after three or four days. The patients are kept under treatment until the urine has been constantly alkaline from ten to twelve days and are then discharged. In only 1 patient was there any complication—a severe necrosis of the mucous membrane of the cheek and severe intestinal hemorrhage.

Treatment of Chronic Protozoic Enterocolitis as Encountered in the Northern United States.—SMITHIES (*Med. and Surg.*, 1917, i, 460) points out that chronic enterocolitis associated with protozoa in the stools is more frequent in the temperate zones than is generally appreciated. In an analysis of 1000 stools examined in the Gastro-enterological Laboratory at the Augustana Hospital, viable protozoa were found in the dejecta of 93 patients. These patients came mainly from the northern half of the Central States. The source of infection was probably water, garden-truck, or fruits contaminated directly or indirectly by protozoa carriers who had at some time lived in the tropics. Chronic enterocolitis associated with protozoa must be recognized as a clinical entity even though its course in the temperate zones is not so severe and dramatic as in the tropics. Thus in the 93 cases studied by Smithies, gastric achylia was present in 43 per cent., subnormal acidity in 36 per cent., and normal or increased acidity in 21 per cent. In 70 per cent. of the cases the stools showed reduced tryptic digestion and in 29 per cent. there was deficient amylolytic ferment. Anemia suggesting the pernicious type was present in 20 per cent. and severe secondary anemia in 67 per cent. of the cases. In 74 per cent. of the cases there was an eosinophilia of 30 per cent. or more. The type of infect-

ing organism must be determined before successful treatment can be instituted and the daily microscopic examination of the stools forms the only control of therapeutic management. In Smithies's series of 93 patients 46 had multiple infection. Treatment consisted in (*a*) measures for freeing the alimentary tract from protozoa; (*b*) management of dyspepsia and malnourishment associated with enterocolitis. Measures for freeing the alimentary tract from protozoa: The patients were put on a liquid diet for two days and given each morning a glass of citrate of magnesia solution. The specific medication depends on the type of infecting organism. In entameba cases the patient is put to bed on liquid diet. To lessen the colic, hot pads moistened in boracic-alcohol solution are applied to the abdomen. For two days the patient receives per ora 10 grains of aluminum salicylate of ipecac and $\frac{1}{3}$ grain of emetin hydrochloride hypodermically every four hours. If the stools show fewer parasites the doses of ipecac and emetin are reduced by one-third, and this is continued for another two-day period. If the parasites are still abundant no reduction is made, but usually by the end of the first week the patient receives emetin $\frac{1}{3}$ grain twice a day and 10 grains of ipecac four times a day. The diet should be liquid or soft. In addition the colon is irrigated with 4 quarts of hot normal salt solution or a solution of quinin (1 to 3000) and thymol (1 to 5000) in normal salt solution morning and night. On the sixth day the patient is put on a fat-free diet for twenty-four hours. On the evening of the seventh day, 30 grains of thymol in honey are given at 8 P.M. and again at 10 P.M. Early next morning he receives 2 ounces of Epsom salts and throughout the morning frequent drinks of black coffee, fat-free broth, or malted milk. During the second week the emetin, ipecac, and bowel irrigations are continued, and on the evening of the tenth day two doses of thymol (15 grains each) are given. By this time the stools are usually free from parasites and the diet may be increased gradually, provided it is low in protein and not bulky. If the parasites persist at the end of two weeks the colon is irrigated with hot normal saline and from 500 to 1000 c.c. of filtered commercial kerosene are given per rectum and retained for at least an hour. If the external parts are coated with carbolated vaseline no harmful effects follow. When flagellate protozoa are the infecting organisms the treatment is much the same as for the entameba, except that no ipecac and emetin are given; instead, evening doses of calomel (5 to 15 grains) are used, followed by 2 ounces of Epsom salts next day. The calomel is repeated about every five days according to the indications furnished by the stool examinations. The management of dyspepsia and malnourishment associated with enterocolitis is treated as follows: When the stools are free from protozoa the enterocolitis is treated by large doses (30 grains) of bismuth subnitrate five times a day. Emetin and ipecac should be continued for at least six weeks and the bowel irrigations for three. The general condition of the patient is improved by the use of iron for the anemia, HCl for the subacidity, etc. The diet consists of cereals, rare meats, simple puddings, milk, and well-washed and cooked vegetables. After-treatment: Smithies believes that the protozoa may lurk in the gall-bladder and the appendix for years, and unless these are removed the patient may become a carrier or be subject to frequent reinfection. When these organs are not removed the stools should be examined at least three times a year.

PEDIATRICS

UNDER THE CHARGE OF

THOMPSON S. WESTCOTT, M.D., AND ALVIN E. SIEGEL, M.D.,

OF PHILADELPHIA.

The Condition of the Reflexes in Poliomyelitis. — REGAN (*Am. Jour. Dis. Children*, November, 1917, xiv, No. 5) states that in poliomyelitis the most common symptom is an alteration in the reflexes accompanying or following a short febrile period. The patellar reflex is the most commonly affected, owing to the frequent involvement of that region of the spinal cord which innervates the quadriceps extensor group of muscles. It was altered in 81 per cent. of 818 cases. Hyperactivity of the knee-jerks is usually, if not invariably, the first change to occur in the preparalytic stages. It may be so marked that the slightest tap with the percussion hammer will elicit a response which will cause the foot to move through the arc of a circle of from 30 to 60 degrees. The reflex, as a rule, becomes diminished and finally absent as paralysis supervenes, but it may persist exaggerated in cases in which the paralytic involvement is slight and in which the lower extremities are not concerned to any marked extent, as well as in cases that are of the spastic variety. It may also be met with when the paralysis is limited to the upper half of the body. In rare cases there may be encountered an absence of knee-jerks on the paralyzed side and an exaggeration on the opposite and healthy side. Hyperactivity of the patellar reflex is most frequent in the meningitic and ataxic cases and in the combined type of the disease in which the meningitic symptoms were prominent. Absent or normal response is the rule in purely bulbar cases except in the very early stages. An exaggerated reflex is sometimes seen in atrophied or obviously paralyzed leg. Not infrequently the knee-jerks are found hyperactive in convalescence when every other symptom of the disease has disappeared, no doubt indicative of the stage of improvement, and just preceding the return to normal. A diminished patellar reflex usually precedes its disappearance with the advent of paralysis. It may, however, persist during the entire course of the acute stage in cases with slight paralysis, especially of the meningitic and ataxic types. During the stage of improvement it is, as a rule, the first change noticed. A sluggish patellar reflex is rather unusual except in the meningeal forms of the disease. Very rarely sluggish but hyperactive reflexes are encountered. Absence of the knee-jerks is the most common alteration, and it is most frequently encountered in the myelitic and bulbar types of the malady. This symptom may be the only demonstrable objective sign of the disease. The patellar reflex may remain normal throughout the entire paralytic phase of the disease, especially in bulbar and ataxic cases. It is also common in myelitic cases when the paralysis is limited to the upper extremities and trunk. It is scarcely ever encountered when the hydrocephalus is at all marked. The plantar reflexes are exaggerated in the preparalytic stage. The hyperactivity is of a peculiar type and is accom-

panied by a response which comprises not only a movement of the foot but also of the entire leg. The reflex was altered in only 41 per cent. of 643 cases during the paralytic stage. An exaggerated response may persist into the paralytic stage, more especially in the meningitic and ataxic classes of cases. A diminished reaction was found most common in the ataxic, the myelitic, and the myelitic meningitic forms of the disease. The reflex was absent most frequently in the purely bulbar and in the bulbar myelitic types. A normal reaction was encountered most often in the ataxic, bulbar myelitic, and bulbar meningitic forms of the malady. Often the first sign of improvement in cases with the marked paralytic involvement is the gradual reappearance of the reflex. There is to be noted two types of response to the movements employed in testing for Kernig's sign, the true reaction and the other, the "pseudo-reaction" or false reaction. The true reaction is only occasionally met with, and is limited to those forms of the disease in which the meningitic symptoms are extremely prominent. The pseudo-reaction is common in all cases exhibiting a moderate or severe degree of polyneuritis, and is associated with the voluntary resistance to the extension of the leg on the thigh, owing to the pain thereby produced. It is a sign analogous to Lasigne's sign in sciatica. The Babinski phenomenon is relatively rare in poliomyelitis, but it occasionally occurs in the meningitic form of the disease. Its presence in a case in which the diagnosis was doubtful would always be decidedly in favor of tuberculosis, and, to a less extent, cerebrospinal meningitis. A true ankle-clonus is likewise very rare in contradistinction to other forms of meningitis. The reaction may be simulated in cases in which the polyneuritis is marked, owing to the severe pain caused by the movements employed. The pupillary reflex is but little altered. It may be slightly sluggish in cases with marked hydrocephalus and during the very early stage; but a reaction is almost always present except in the moribund stage. When paralysis is limited to one leg only the knee-jerks on the healthy side are more often present than absent. When the upper extremities are alone involved the patellar reflexes are more often present than absent. In a small percentage of cases of facial nerve paralysis the knee-jerks may be lost.

Infantile Scurvy: A Study of its Pathogenesis.—Hess (*Am. Jour. Dis. Children*, November, 1917, xiv, No. 5) says one of the several factors in the pathogenesis of infantile scurvy is faulty diet. In his studies pasteurized milk was found to be a contributing cause if it was not fresh; that is, if given twenty-four to forty-eight hours after pasteurization. From this point of view milk pasteurized in the city was preferable to that pasteurized at the creamery, which reaches the consumer much longer after the heating process. Aging seemed to play a greater role in the production of scurvy than heating, whether the milk was pasteurized or raised to the boiling-point. It was found that even raw milk on aging lost its antiscorbutic properties. Infantile scurvy is not a simple dietary disease. The diet is at fault in allowing the intestinal bacteria to elaborate toxins. The toxin is not always the same, and perhaps it would be better not to regard this condition as a clinical entity. Scurvy is an intestinal intoxication or auto-intoxication, due to the overgrowth of harmful bacteria in the intestines.

It is the product of an unbalanced flora which is no longer controlled by the proper dietary. Oliguria is a common symptom. The mild therapeutic effect of citric acid may be attributed to its diuretic properties. Orange-juice was found to bring about marked diuresis. One of the striking and important symptoms of scurvy is a susceptibility to infection, such as furunculosis, nasal diphtheria, "grippe," etc. Some hemorrhages are due to this secondary infection, and are to be regarded not as scorbutic but rather as focal complications. Other hemorrhages are truly scorbutic. Scurvy is essentially a disorder characterized by malnutrition and not by hemorrhages, taking months to develop, and from a clinical point of view frequently latent or subacute. Scurvy occurring in epidemics is described. This results when latent scurvy exists among a number of infants, and is associated with an infectious disease such as "grippe."

The Significance of Certain Dental Stigmata of Congenital Syphilis.—WALL (*Arch. Pediat.*, October, 1917, xxxiv, No. 10) states that stigmata of inherited syphilis is evidenced by abnormalities of the permanent teeth. The virus of syphilis affecting the fetus or infant in the early months of life finds a habitat sufficiently pliable to afford a fertile field for destructive tissue change and for an equally deleterious inhibitory effect upon the tender formative structure within the aveolar processes. If the luetic poison has been in the ascendancy during certain stages of the development of the permanent teeth, especially that of dentification, the child will in later years show certain changes, such as were first brought to light by Hutchinson. This lesion of the central incisors bears his name. Other teeth as well show the stigmata of inherited syphilis, and there are other teeth which show characteristic lesions. The first permanent molar or sixth year molar presents typical deviation from the normal if the patient shows dental stigmata at all. The three dental lesions seen are Hutchinson's teeth, cuspal erosion of the first permanent molar, and dystrophies of the permanent teeth. The sixth year molar is the only permanent tooth to dentinify during intra-uterine life. This occurs about the fifth to sixth month. The changes in this tooth are characteristic. There is an erosion of the grinding surface. It is irregular in form. The surface of the tooth is pitted, excavated and discolored. Often a pulpy mass of a dirty yellow color occupies most of the surface of the crown. It is sometimes called "the honey-combed molar of hereditary syphilis."

The Relation of the Reaction of the Urine to the Diet in Infants and Children.—TORRES (*Am. Jour. Dis. Children*, November, 1917, xiv, No. 5). In changing the reaction of the urine in children the difficulties are diminished when a suitable diet is prescribed. By diminishing the amount of protein to 2 grams and sometimes to 3 grams per kilo the urine will often be rendered alkaline without using a drug. In cases in which the protein reaches the lowest physiological limit without rendering the urine alkaline the acidity may be diminished by adding vegetables to the diet. In older children in whom the food is more varied, much can be done by regulating the diet. The urine of carnivora is acid and that of herbivora is alkaline. The acidity in the carnivorous animals depends on the products of protein

metabolism. In cases in which vegetables are used to turn the urine alkaline, vegetables must be used in which the amount of protein is smaller in proportion to the salts, like fruits, rice, wheat and the like, rather than those in which a large amount of protein is present, such as peas, beans and the like. Vegetables which contain purins like coffee, tea and cocoa may increase the acidity, because they may produce uric acid. In cases of infection, especially of the urinary tract, and in cases of starvation, the urine tends to become more acid than in other conditions; therefore it is more difficult to render it alkaline. When drugs are used without the proper diet, the dose necessary to make the urine alkaline is much larger than is usual. The action of the drugs is very fleeting, and should be watched and given in larger and more frequent doses as necessary.

OBSTETRICS

UNDER THE CHARGE OF

EDWARD P. DAVIS, A.M., M.D.,

PROFESSOR OF OBSTETRICS IN THE JEFFERSON MEDICAL COLLEGE, PHILADELPHIA.

The Lower Uterine Segment. — In a discussion before the Royal Academy of Medicine in Ireland, TWEEDY (*Jour. Obst. and Gynec. Brit. Emp.*, June–August, 1915) stated that there is now sufficient data to clear up the mystery of the anatomy of this part of the womb and the endoperitoneal tissue is the true boundary between the cervix and body, and constitutes the tendinous extremities of the uterine fibers. Until this tissue has been made inert by rupture or by opening of the internal os, no direct pressure can be brought to bear on the cervix. In fact, dilatation of the internal os is an early phenomenon in pregnancy which permits the ovum to pass through and press directly on the cervix. A continuous pressure causes a rapid hypertrophy in the cervix which does not stretch but grows.

Intestinal Hemorrhage in the Newborn.—CRAWFORD (*Jour. Obst. and Gynec. Brit. Emp.*, June–August, 1915) describes 2 cases of intestinal hemorrhage in female infants, the bleeding appearing thirty-six and fifty-two hours after birth. The labors had been normal. In the first case horse serum was given subcutaneously in three doses of 2 c.c., and with each a small dose of adrenalin. This infant died on the fifth day and a duodenal ulcer was found very near the pylorus. The second case was treated by antidiphtheric serum, two doses of 5 c.c. each, followed by recovery. Breast-feeding was resumed without difficulty on the fifth day. The only symptom in both cases was free hemorrhage from the rectum and vomiting was absent. In discussion a case was described of an infant three days old that died three hours after having nose-bleeding.

Gunshot Wounds of the Body During Pregnancy.—SMEAD (*Am. Jour. Obst.*, December, 1916) describes the case of a patient, aged twenty-five years, pregnant, at term, who was accidentally shot in the back. The bullet entered about one inch below the twelfth rib on the right side at the outer edge of the quadratus lumborum and could be felt lying under the skin of the abdomen about two inches below the heart and to the right of the umbilicus. The patient was poorly nourished, heart and lungs normal, and urine free from albumin. There was no elevation of temperature on admission. The abdomen was hard, tense, very sensitive and slightly distended. A small quantity of blood was escaping from the wound in the back. The child's heart was strong and nearly normal in rate. Upon opening the abdomen, the peritoneal cavity contained a large quantity of free blood and coagula with which amniotic liquid was mixed. There was a perforation of the posterior wall of the uterus to the right of the midline about three inches below the fundus, and a second was present on the anterior wall about two inches below the fundus. Between the two openings the course of the bullet was about five inches. The size of the uterus made it impossible to properly explore the abdomen for intestinal perforations, and, accordingly, Cesarean section was immediately done. The placenta had been perforated by the bullet. The child was readily delivered and cried immediately. Its only injury was that the ring finger on the left hand had been broken and lacerated by the bullet. The uterus contracted normally. The uterine incision and the bullet wound were closed with chromic catgut. When the blood was sponged out of the abdomen the intestines were examined for perforations. The bullet had entered between the folds of the mesentery of the ascending colon and passed through the bowel, making two perforations. It had then gone through the uterus and into the abdominal wall without injuring the small intestine or any other organs. The perforations in the colon were closed and a drain was passed down to that on the posterior surface of the bowel. The abdomen was drained by three soft tubes; the mother made an uninterrupted recovery. The child was nursed and developed normally. The broken finger was pieced together and healed by first intention, although there was a very slight deformity. The writer has collected 29 cases, showing a surprisingly low mortality if operation is promptly performed. In discussion, Davis reported a case of a woman, pregnant three and a half months, accidentally shot by a small rifle, the bullet making twenty-one perforations in the abdominal organs. She was brought by train 85 miles to hospital and was seen twelve hours after the receipt of the injury. Five feet of intestine were removed, including nineteen perforations. The patient recovered and was delivered of a living child at the ninth month.

Pelvic Infections in Pregnancy.—MOORE (*Am. Jour. Obs.*, November, 1916) describes a peculiar type of pelvic infection complicating pregnacy. His patient was a rachitis negress who was delivered by Cesarean section of a living, full-term child and suffered from puerperal neuritis while in the hospital. This appeared in both arms, in the posterior tibial muscles and fingers. The patient again came into the hospital and was delivered spontaneously of a premature child which did not long survive. The patient was ill-nourished, with slightly

enlarged thyroids, and stated that there had probably been a premature rupture of the membranes and hemorrhage before she entered the hospital. Two hours after her delivery the patient had a severe chill, the temperature rising to 105° F. At three o'clock the next morning she had another chill, temperature rising to 104° F., after which it gradually declined to normal during the next twenty-four hours. The blood showed a pure growth of Staphylococcus aureus. The leukocytes were 11,200. On the seventh day after delivery the blood was negative but lochial discharge showed many colonies of Staphylococcus aureus. The patient had tenderness over the entire tibial muscles which appeared to be neuritis of a mild type. The lochia was not foul and the patient felt well during her illness.

Dermoid Cyst of the Ovary with Twisted Pedicle and Acute Appendicitis Complicating Pregnancy.—DOYLE (*Am. Jour. Obst.*, November, 1916) reports the case of a primipara who had had abdominal pain at various periods of her life before marriage. Menstruation had ceased about four and a half months previously. The patient then had an attack of sharp, sudden pain in the right lower abdomen with vomiting and prostration. This lasted three days. One week later the pain again appeared on the right side and was more severe. To the right of McBurney's point and lower there was a mass of considerable size, very tender and painful. Vaginal examination was unsatisfactory. At operation an ovarian tumor was found, with two twists in its pedicle, and behind it an acutely inflamed appendix, adherent to the posterior wall of the pelvis. Both tumor and appendix were removed, the patient making an uninterrupted recovery. Pregnancy was not disturbed, and the patient ultimately gave birth to a healthy, normal child. The tumor was a dermoid filled with sebaceous material, and contained six teeth and some hair.

Myomectomy in Pregnancy.—ALFIERI (*Anal. di Ostetr*, No. 8, 1916) reviews the literature of the subject and believes that in performing myomectomy during pregnancy the operator must be sure that the substance of the uterus is not essentially damaged and that the uterus is left in such a condition as to safely perform the function of parturition. While it is desirable to practice conservatism, this must not be carried to the extent of retaining a tumor whose presence might seriously complicate labor.

Management of Tumors Complicating Pregnancy, Labor and the Puerperal State.—BEACH (*Am. Jour. Obst.*, June, 1916) believes that it is of the utmost importance that the presence of ovarian tumor complicating pregnancy be early diagnosticated. The smaller the tumor the more apt it is to have torsion of the pedicle followed by gangrene. The occurrence of some acute intra-abdominal calamity during pregnancy should always suggest the possibility of a small ovarian tumor which has undergone torsion. Rupture and suppuration of such tumors may occur. As regards treatment, statistics abundantly show that operation is far safer than the expectant method. When tumors are discovered during the first half of pregnancy, unquestionably the safest

method of treatment is removal. The abdominal operation is far safer than the vaginal. When ovarian tumors are discovered during the second half of pregnancy, if the patient is at term, Cesarean section should precede or follow removal of the tumor. The writer describes the case of a girl, aged eighteen years, in her second labor, in whom it was thought there was an ovarian cyst behind the uterus complicating labor. The child was living and uterus showing high retraction ring. An attempt to replace the tumor under anesthesia failed. Upon opening the abdomen the lower segment was greatly thinned and the pedicle of a right-sided ovarian tumor passed down posteriorly behind the head into the pelvis. The tumor could be felt along the side of the head. An attempt was made to draw the tumor out of the pelvis while an assistant pushed the head up from below. This was unsuccessful and the pedicle of the tumor began to tear. With considerable difficulty a trocar was inserted into the tumor and a small quantity of mucilaginous substance was discharged. The operator then made traction on the head upward by the hand in the abdomen while an assistant made pressure on the tumor from below. This was successful. The head was then pressed down and delivered by forceps from below, followed by removal of the tumor through the abdomen. The placenta was expressed by the hand within the abdomen and the abdomen closed. Both mother and child made a good recovery. In cases of ovarian tumor complicating pregnancy and labor in which the tumor is adherent, infected or broad ligament tumor or when there is any grade of pelvic contraction, Cesarean section is the operation of choice. The tumor is dealt with after the removal of the child. In infected cases which develop late in labor, abdominal section should be at once performed. In cases in which the presence of the tumor is first discovered during the puerperal period, operation still remains the safest method of procedure.

Induction of Labor in Normal Pelvis at Term.—REED (*Surg., Gynec., and Obst.*, March, 1916) argues that because the physiological duration of human pregnancy is not exactly known, and the factors that determine the onset of labor are exceedingly obscure, that one should not allow the mother to delay in pregnancy after full term has been reached. The dangers of prolonged pregnancy are overgrowth of the child, injury to mother and child, with chance of death of the fetus and considerable risk for the mother. He takes the length of the fetus as normally 50 cm. and the weight between 5 and 8 pounds. He measures the length of the child *in utero*, using a pelvimeter from the upper border of the symphysis to the breech of the child. This measurement is doubled and 2 cm. subtracted for the thickness of the abdominal wall. The result of this calculation should give the length of the child. In his cases this measurement had rarely varied more than 2 cm. from the actual length of the child obtained at birth. Müller's method of crowding the head into the pelvis may also be used to ascertain the comparative size of the pelvis and fetus. These measurements are also correct by a careful computation of the length of pregnancy taken from the menstrual history. The induction of labor is brought about after antiseptic precautions by introducing a Voorhees bag without rupture of the membranes. The bag is introduced and moderately distended, and if pains do not start

within a hour then a weight of 1 or 2 pounds is attached by a tape to the protruding tape and passed over the foot of the bed. Usually in from five minutes to half an hour contractions begin and generally go on to spontaneous expulsion. Two objections are against this procedure: One, the possibility of infection, second, the fact that a mistake in calculation may be made and the child delivered before it is thoroughly viable. Again, the dilating bag may break; 100 consecutive cases were treated in this way, 35 primiparæ and 65 multiparæ. The average duration of labor was seven hours and forty-five minutes; the shortest labor fifty-five minutes; the longest thirty hours. During, or shortly after, the insertion the bag broke six times and was reinserted three times. The average time for the expulsion of the bag was three hours and twenty minutes. The membranes were ruptured by the introduction of the bag twice. There were two maternal deaths: 1 from placenta previa with myocarditis and 1 from pneumonia eight days after labor. The average weight of the child was 7 pounds 7 ounces; the smallest 5 pounds; the heaviest 10 pounds 5 ounces. Seven children died. There were 3 cases of version and extraction; 17 cases of laceration of the perineum; forceps was used in 23 cases. In 7 patients there was postpartum rise of temperature. In but 1 case, however, was there tenderness over or beside the uterus or foul discharge or subinvolution. In only 1 case was it necessary to use the catheter after delivery. While this proposition seems attractive it cannot be endorsed by conservative obstetricians. The dilating bag often causes severe suffering, while the risk to the mother and child are greater than in spontaneous labor. This procedure disregards the natural preparation of the child for labor by its descent into the pelvic cavity. A much better criterion than the attempted measurement of the child *in utero* consists in watching the development of pregnancy, and, in primiparæ, studying the descent of the child's head into the pelvic cavity. When the patient comes to term, and this does not occur, there is some abnormality which may require the bringing on of labor. In multiparæ it is best to calculate, as accurately as possible, the period of pregnancy, and the former experience of the patient is always available for the guidance of the physician.

Cesarean Section for Placenta Previa.—KRÖNIG (*Deutsch. med. Wchnschr.*, 1916, 1178, xlii), reasoning on the accepted clinical principle that not only the mother but the child must be saved, believes that version and the use of the dilating bag cannot be considered satisfactory in placenta previa. Both are exceedingly dangerous to mother and child. When the ovum is of low attachment the wall of the lower segment is infiltrated with fetal cells which sometimes cause rupture. The stretched condition of this portion of the uterus renders such operations as vaginal Cesarean section, version or dilatation, with instruments or a bag, very undesirable. When a patient with placenta previa has fever and has been tamponed on account of severe hemorrhage it may be difficult to determine just which method of Cesarean section should be chosen. Section through the body of the uterus is contra-indicated if the os is larger than a silver dollar.

PATHOLOGY AND BACTERIOLOGY

UNDER THE CHARGE OF

OSKAR KLOTZ, M.D., C.M.,

PROFESSOR OF PATHOLOGY AND BACTERIOLOGY, UNIVERSITY OF PITTSBURGH, PITTSBURGH, PA.

Movements of Tissues in Culture Media. — LOEB and FLEISHER (*Jour. Med. Res.*, 1917, xxxvii, 75) give a resumé of their previous work and include some new experiments in this significant problem, which, being merely in its infancy, permits of but provisional conclusions. The authors consider three factors of importance in determining the movement of tissue cells which are brought in contact with culture media: (1) a factor recognizable for the initial sending out of pseudopodia, the primary desequilibrizing factor; (2) the contact with solid bodies which strongly influence the direction in which the cells move; (3) a centrifugal force driving the cells into the coagulum and away from the tissues. In other words, an innate centrifugal tendency and an outside factor, the stimulating action of contact with a solid body. This mode of reaction they have called sterotropism, which is based on surface changes occurring in cells under the influence of variations in the surrounding medium. Such modifications in the cell surface lead to a viscid condition of the ectoplasm, causing it to stick to solid bodies, thus causing the initial desequilibrization as well as the movements in contact with foreign bodies, such as threads. It may be expected that the stimulus calling forth the sending out of pseudopodia affects especially that side of the cells which adjoins the coagulum (of blood plasma) while the other side adjoining the tissue is less affected. This condition itself would tend to lead to a movement in the direction of the coagulum. As to the centrifugal movement of cells away from their tissue base into the coagulum it is probable that the principal factor is the negative chematropic influence which living cells exert upon one another together with the positive attraction for the cells, exerted by certain constituents of a clot or necrotic materials, and also the mechanical factors as of contact and the polarized change in the environment of the cells. The presence of dead tissue is the factor that directly or indirectly determines the direction of movements of various kinds of cells, but this stimulus is absent under ordinary conditions in the body. Connective-tissue cells, and to a less extent differentiated epithelial cells and carcinoma, move actively in a coagulum *in vitro*, but not in the normal organism. Epithelial cells from the guinea-pig in a culture medium of coagulated blood serum were seen to grow and to divide mitotically without any association with connective tissue, behaving essentially like ameba and moving less freely than connective-tissue cells. The growth of the latter, on account of their importance in the formation of scar tissue, is of special interest. There are many factors which influence their growth into the coagulum. For this work the authors used pieces of rabbit kidney into which incisions had been made five to eleven days previous to the experiment in order to increase

the number of fibroblasts. The degree of contact between coagulum and tissue is of importance, since the fibroblasts do not grow across a space such as may be formed by the action of fibrolysins. The ingrowth of cells is more rapid if the fibers of the coagulum are parallel to those of the tissue, and the degree of hardness and the character of the surface of the coagulum are of significance; the immigration is the more difficult the denser the coagulum and the smoother its surface. The fibroblasts travel by sending out pseudopodia along the lines of the fibrils of the coagulum. The authors noticed among the fibroblasts certain ones which seemed to have a phagocytic function. As to the movement of other tissue cells in the coagulum, that of kidney tubule cells and of mouse carcinoma cells is slight. In most of these experiments growth was at its height three days after placing the tissues into the culture media; after seven days all the cells in coagulum and media were necrotic.

The Incidence of Bovine Infection of Tuberculosis in Man.—Careful analyses of the infecting microörganism in tuberculosis of man are relatively few. We have gradually had it impressed upon us that the tubercle bacillus present in various tissue lesions was not always of the same kind. The attitude of Koch, in 1901, gave the impression that we need look for only one type of bacillus, the human, in studying the nature of the infective microörganism in pulmonary lesions. Newer methods of technic have given us an opportunity of studying the tubercle bacillus with greater ease, and have also opened the way for distinguishing the various types of these organisms and demonstrating their presence in human lesions. With these newer methods at hand, WANG (*Jour. Path. and Bact.*, 1917, xxi, 131) has isolated tubercle bacilli from a variety of sources. The organisms were then studied and tested to determine the type. Of 123 strains of tubercle bacilli obtained from 88 cases of tuberculosis in man, all conformed with the regular human or bovine type. Bovine bacilli were isolated 7 times in an examination of sixty-eight adults and the same microörganism was obtained 11 times from twenty children. These bovine bacilli were found in the sputum, lymph glands, abdomen, and meninges. The high percentage (55) of bovine infection in children under sixteen is striking. The author believed that the distribution of lesions as found at autopsy indicated the intestinal route of infection in 50 per cent. of the children, while in adults it fell to 13 per cent. In studying other analyses of the incidence of bovine infection in Edinburgh the author finds that in a total of 281 cases in which the type of tubercle bacillus was determined, 78 per cent. in children under five, 70 per cent. in children from five to sixteen, and 7.8 per cent. in adults over sixteen were of bovine type. The author was also able to show that tuberculous processes which had undergone calcareous change still retained some of the original infection.

Spirocheta Morsus Muris the Cause of Rat-bite Fever.—Much interest has recently been shown in studies upon rat-bite fever. In all there have been over 80 cases of human infection reported in the literature. The disease in man has definite clinical manifestations, among which the periodic recurrence of the fever, swelling of lymph glands and the appearance of a skin rash are the most important. There is

usually an incubation period of from ten to twenty-two days. Schottmüller reported the finding of a streptothrix in the enlarged lymph glands of a patient, and this was subsequently confirmed by Blake, who was able to cultivate the microörganism. Shortly following this, Tunnicliff reported upon the finding of a similar streptothrix in the lungs of rats suffering from chronic bronchopneumonia. Her findings suggested that the rat was subject to spontaneous infection by this microörganism, and that during the infectious stage was capable of transferring infections to man. Shortly following the report by Blake, a brief report from Japan claimed the finding of a spirochete in the blood and tissues of 2 patients with rat-bite fever. These same authors, FUTAKI, TAKAKI, TANIGUCHI and OSUMI (*Jour. Exper. Med.*, 1917, xxv, 33) bring further evidence of the specific nature of a spirochete for this disease. They have had the opportunity of studying 5 more cases, in each of which the spirochete has been demonstrated and in 2 the microörganism was found in the circulating blood. The authors clearly point out that the disease under discussion is one having clinical manifestations as detailed above. It is pointed out that the rat may serve as a carrier for a variety of microörganisms pathogenic to man, and that the various infections may be transmitted through bites. The spirochete which they have found is larger than the Spirocheta pallida, and, furthermore, possesses a flagellum. With difficulty the microorganism is demonstrated directly in the lesions of man, but is more easily shown when infected tissues are inoculated into mice. Mice, rats, guinea-pigs and monkeys may all be infected. In the monkey a disease may be induced having some of the manifestations observed in the human cases. The spirochete has been successfully cultivated. Associated with this work, ISHIWARA, OHTAWARA and TAMURA (*Jour. Exper. Med.*, 1917, xxv, 45) carried on a series of animal experiments. Eighty rats were caught and were made to bite guinea-pigs. Of this number, 10 produced experimental rat-bite fever in the exposed animals. They have been unable to detect the spirochete in the mouth of the infected rats. The clinical course of the disease in the guinea-pig resembled that in man, though the progress of the fever was not as regular. The disease could be propagated to new animals by the inoculation of blood or tissue emulsions obtained from the originally infected guinea-pigs. Upon infecting rats or mice, the spirochete could be demonstrated in the peripheral blood. These authors are not as yet convinced that the spirochete which they have demonstrated is identical with that isolated by Futaki and his co-workers. The spirochetes of Ishiwara is short and appears to possess a flagellum at each end. The spirochetes disappear from the blood of infected animals by the use of salvarsan.

Fistula in Ano and Tuberculosis.—The causal relationship between fistula and tuberculosis has been a much discussed question. STONE (*Am. Rev. Tuberculosis*, 1917, i, 548) quotes Melchior as finding 61 per cent. of his fistulæ tuberculous, whereas Frey found a similar relationship in only 6.9 per cent. of 72 cases; the latter relied entirely upon a microscopic finding of a typical lesion, but the former, in addition to this, bases his figures on a diagnosis of tuberculosis elsewhere, usually in the lungs, present before, after or at the time of the fistula. Of 123

cases seen by the author, 31 were carefully examined for tuberculosis; 13 presented active lesions and 7 showed evidence of previous involvement of the lungs. Microscopic examination of tissue from 10 fistulæ, all but 2 of which were from the groups which had either active or inactive tuberculosis, showed only one containing tubercles. Nevertheless, from his own experience and from that of others, he feels that there is a causal relationship between fistula and tuberculosis in 15 per cent. to 30 per cent. of the cases, while the characteristic lesion in the fistula can be demonstrated in only about 10 per cent. A corollary to this is the author's final conclusion that in a considerable percentage of cases no relationship exists between fistula in ano and tuberculosis.

Contributions to the Biochemistry of Pathogenic Anaërobes.—Starting with the assumption that the effect of anaërobes is mainly local and the changes brought about lend themselves readily to analytical study, WOLF and HARRIS (*Jour. Path. and Bact.*, 1917, xxi, 386) have thrown much light on the biochemistry of B. welchii and B. sporogenes. These two anaërobes serve as examples of the saccharolytic and proteolytic groups of Henry. The authors made careful investigations on gas production, ammonia and amino-acid formation, production in tritratable acidity as well change in hydrogen-ion concentration and the sugar consumption of these bacteria with a variety of media. The methods employed are relatively simple. Different strains of the two anaërobes were used in practically all of the experiments which make it somewhat difficult to compare the various results. The results obtained are important. B. welchii in a medium containing 3 or 4 per cent. of lactose may give a volume of gas 3.8 times that of the medium used. Gas produced in sugar-free media such as casein is difficult to explain and the authors suggest that it may be due to the production of amino-acids which may be glycogenetic. In the experiments with peptone water prepared from casein the amino-acids appear to have provided the pabulum for gas production. The velocity of gas production shows a latent period, a sharp rise and then a secondary rise. The composition of the gas first formed was on one occasion 90 per cent. hydrogen while the relationship of hydrogen to carbon dioxide in the final composition was roughly 2.1. The activity of growth of B. welchii and its death are closely associated with the hydrogen-ion concentration, and it would appear that the optimum is at or below P_H=4.9, and the lethal point between P_H=4.52 to P_H = 4.56. The importance of the amino-acids is indicated by the increase in these substances before gas production took place. Volatile acids made up about 60 per cent. of the total acidity, and of this nearly all was normal butyric acid. The B. sporogenes shows many marked differences. The amount of gas produced is almost as much as that by B. welchii, and it produces it in practically all media. The amount of gas formed by B. sporogenes is the same in 4 per cent. alkaline casein and sugar in addition. The B. sporogenes takes longer to establish itself and produce gas on many of the media than does B. welchii, but during this period it is very active in amino-acid production. The proteolytic enzymes of this anaërobe attack media in which extensive proteolysis has already taken place. Ammonia was formed in one experiment

in quantities to more than balance the original amino-acids present in the medium (peptone water). In alkaline casein 20 per cent. of the total nitrogen originally present is transformed into ammonia. The strongly basic properties of ammonia are important. Butyric acid is formed by B. sporogenes as well as other organic acids. In conclusion, the author suggests the possibility of using a solution, for treatment of wounds with B. welchii present, in which the hydrogen-ion concentration is $P_H = 4.5$. The article is filled with suggestions for much future work and will stimulate biochemical investigations of bacterial activity.

HYGIENE AND PUBLIC HEALTH

UNDER THE CHARGE OF

MILTON J. ROSENAU, M.D.,

PROFESSOR OF PREVENTIVE MEDICINE AND HYGIENE, HARVARD MEDICAL SCHOOL, BOSTON, MASSACHUSETTS,

AND

GEORGE W. McCOY, M.D.,

DIRECTOR OF HYGIENIC LABORATORY, UNITED STATES PUBLIC HEALTH SERVICE, WASHINGTON, D. C.

Pneumonia.—A great advance in our knowledge of pneumonia has recently been achieved through the work of COLE (*Arch. Int. Med.*, 1914, xiv, 56), DOCHEZ and GILLESPIE (*Jour. Am. Med. Assn.*, 1913, lxi, 727), HANES (*Jour. Exper. Med.*, 1914, xix, 38), AVERY (*Jour. Exper. Med.*, 1915, xxii, 105). Four groups or strains of pneumococci are now recognized: (1) being all diplococci they can be differentiated only by their agglutination reactions with specific sera, except Group III, which is morphologically characteristic. Group I is the cause of from 30 to 47 per cent. of all cases of pneumonia and has a mortality of about 24 per cent. Group II is indistinguishable from Group I save by the agglutination reaction. It is found in from 18 to 39 per cent. of cases and is fatal in about 60 per cent. of cases caused by it. Immune sera (2) produced by each of these two groups have been obtained from horses and seem to have specific therapeutic value. Group III is the Pneumococcus mucosus (3) formerly classified with the Streptococcus mucosus. It has a distinct mucoid capsule and produces an abundant, stringy mucous growth on surface colonies. It is the cause of from 8 to 13 per cent. of all pneumonia and is fatal in about 61 per cent. of cases. Group IV comprises a heterogenous lot of pneumococci not classified with Groups I, II or III. Each strain of cocci in this group produces a specific agglutinin for itself, which does not agglutinate the other strains or groups. This is the type ordinarily found in normal mouths. These organisms are of low virulence, cause about 20 per cent. of cases, and have a mortality of only about 7 per cent. These four groups seem quite stable and show no tendency to mutation. The pneumococci of the first three groups cause about 80 per cent. of all cases of pneu-

monia and occur only in the mouths of people recovering from the disease or of those in direct contact with such cases. In other words, pneumonia is a contact disease in at least 80 per cent. of cases, the infection being obtained from a patient or carrier.

Infant Mortality.—HIBBS (*Quarterly Publications of the American Statistical Association*, December, 1915) studied the relation of infant mortality to social and industrial conditions, its recent decline in the United States, the influence of prenatal conditions and the size of the family, the relation of the mother, the effect of urban, housing, and living conditions, and the influence of economical and industrial conditions. He concluded that the fundamental cause of the excessive rate of infant mortality in industrial communities is poverty, inadequate incomes, and low standards of living, with their attendant evils, including the gainful employment of mothers. The employment of the mother in gainful occupations is simply the remedy for these evils or "adverse conditions" which the working people in industrial communities have adopted. Undoubtedly, this recourse has had an important effect on the problem, in many cases actually tending to reduce the rate of infant mortality, while in others having just the opposite effect. The primary question in considering the social causes of infant mortality is whether the employment of mothers and married women in extradomestic occupations is, from the view-point of society, as a whole, a good remedy for poverty and an acceptable means of mitigating its influence on the health and mortality of babies and young children. From the point of view of the individual or poverty stricken family the fact cannot be escaped that this effect may be both good and bad: bad, in that it causes the baby to be artificially fed, forces the mother to be absent from home, and in other ways lowers her efficiency as a mother; good, in that it increases the family income and decreases the influence of poverty. We are thus forced to conclude that the fundamental, economical and industrial factor of infant mortality is low wages. The fundamental remedy is obviously higher wages. Other remedies, such as legislation restricting or regulating the employment of mothers before and after confinement, day nurseries, the instruction of mothers and school girls in domestic economy and the like, all have their place; but the chief thing remains the provision of an adequate family income.

A Cheap and Efficient Liquid Disinfectant.—A. F. STEVENSON (*U. S. Public Health Reports*, October 8, 1915) gives a method for the preparation of a new and efficient liquid disinfectant, and suggests as a name for it the "hygienic laboratory Pine Oil Disinfectant." The disinfectant is prepared from pine oil obtained by the steam or solvent process, and is emulsified with a saponified rosin in accordance with the formula given in the article referred to. Standardization experiments with a large series of tests made on different batches of the preparation showed it to have a Hygienic Laboratory phenol coefficient of from 4 to 6. The material can be prepared without the use of special apparatus. The pine oil and rosin are heated together in a covered enamelware pail until the rosin is dissolved. The mixture is cooled to 80° C., sodium hydroxide solution added, and the mixture violently

stirred for about ten minutes; sufficient water is then added to bring the mixture to the original weight. It is then quickly cooled and stored in glass or metal containers until wanted. The finished product is of a dark reddish-brown color, is rather thick and oily in appearance, free from turbidity and cloudiness, and has rather a pleasant odor, somewhat similar to turpentine. When added to water it forms a milky emulsion. Tests showed that the germicidal powers of the disinfectant remain practically constant for about two months, after which time, however, a noticeable deterioration occurs. The author claims that the disinfectant may be used wherever coal-tar compounds are used, and on account of the difficulty of obtaining upon the market the crude products for the production of coal-tar compounds, he believes that this disinfectant will come into general use.

The Sanitary Conditions of Hotels.—Dr. Leverett D. Bristol, Commissioner of Health of the State of Maine, announces the passage of Rules and Regulations of the State Department of Health Governing the Sanitary Conditions of Hotels. This act, approved December 31, 1917, marks Maine the first State to take legal action of a comprehensive nature in this important sanitary problem. The rules are as follows:

1. *General Cleanliness.* No garbage, manure, filth, decaying or other organic material which may serve as a breeding-place for flies or harbor the agents of infectious diseases shall be left exposed or allowed to accumulate upon any hotel or restaurant lot. For those hotels which are not in close proximity to other buildings the term lot shall be understood to include the area within 100 yards of the hotel buildings.

2. *Food.* No food shall be left exposed to dirt, flies, rats, mice, water-bugs or other vermin between such times as meals are being served or at any other time.

3. *Cold Storage.* All refrigerators or rooms in which food is kept in cold storage shall be clean and free from decaying food at all times. Such refrigerators and store-rooms shall be kept at a temperature below 50° F.

4. *Screening.* The doors and windows of all hotel and restaurant kitchens, dining-rooms and rooms where food is prepared or kept shall be adequately screened against house flies and other insects.

5. *Garbage Disposal.* All garbage and kitchen waste shall be kept in covered metal containers or otherwise adequately protected from access to flies and rats.

6. *Infectious Food Handlers.* No person having a communicable disease shall be allowed to handle or serve food. If an employee of a hotel or restaurant is suspected to be harboring the organism of any infectious disease the manager shall require such person to submit to him a certificate of health from a reputable physician or shall notify the State Department of Health by telegram of existing conditions.

7. *Washing Hands after Using Toilet.* All food handlers are required to wash their hands directly after using the toilet, water-closet or urinal, and a notice to this effect, furnished by the State Department of Health, shall be posted conspicuously in each toilet used by such persons.

8. *Clean Rooms.* All hotels shall be kept clean, in a sanitary condition, and free from noxious odors arising from any sewer, drain, privy or other source within the control of the owner, manager or agent.

9. *Clean Linen.* Every hotel shall provide each bed, bunk, cot or other sleeping place for the use of guests with clean sheets and pillow cases; and in no case after being used by one guest shall such sheets and pillow cases be used for another guest until they have been thoroughly washed and dried.

10. *Safe Water Supply.* No hotel or restaurant shall furnish to their guests water which is polluted with human or animal excrement.

11. *Analyses of Private Water Supplies.* Any private water supply in use by a hotel shall be analyzed by the State Department of Health at least once in two years and at such other times as may be required by the department, it being understood that no expense beyond express charges shall be borne by the hotel proprietor. Such water supplies shall at all times be subject to inspection.

12. *Sanitary Privies.* All privies shall be made fly-tight and kept in such sanitary condition as may be approved by the State Department of Health.

13. *Hotels.* Every hotel shall comply with the local and State laws or regulations regarding the disposal of sewage and the use of individual towels and drinking cups.

14. *Windows.* No windows in a bedroom or other public room shall be so fastened that they may not be opened to provide necessary ventilation if no other means of proper ventilation are provided. Where storm-windows are installed some provision must be made whereby access to fresh air may be easily obtained.

15. *New Management.* Whenever a hotel or restaurant changes management it shall be the duty of the new manager to notify the State Department of Health of such change.

16. *Certificates.* Upon receipt of $3 from the management of any hotel or restaurant complying with these regulations the State Department of Health may issue a revocable certificate, expiring one year from the date on which it is issued and having the date of expiration plainly printed thereon. The certificate may be renewed upon the payment of the above-mentioned fee.

17. *Regulations Shall be Posted.* A copy of these rules and regulations shall be posted in conspicuous places where they will be frequently seen by all employees at each hotel or restaurant.

(EDITOR'S NOTE.—A rule requiring the washing of dishes in scalding hot water after each use has important hygienic value.)

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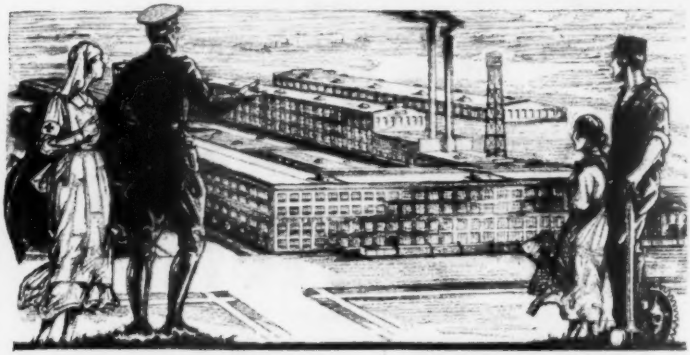

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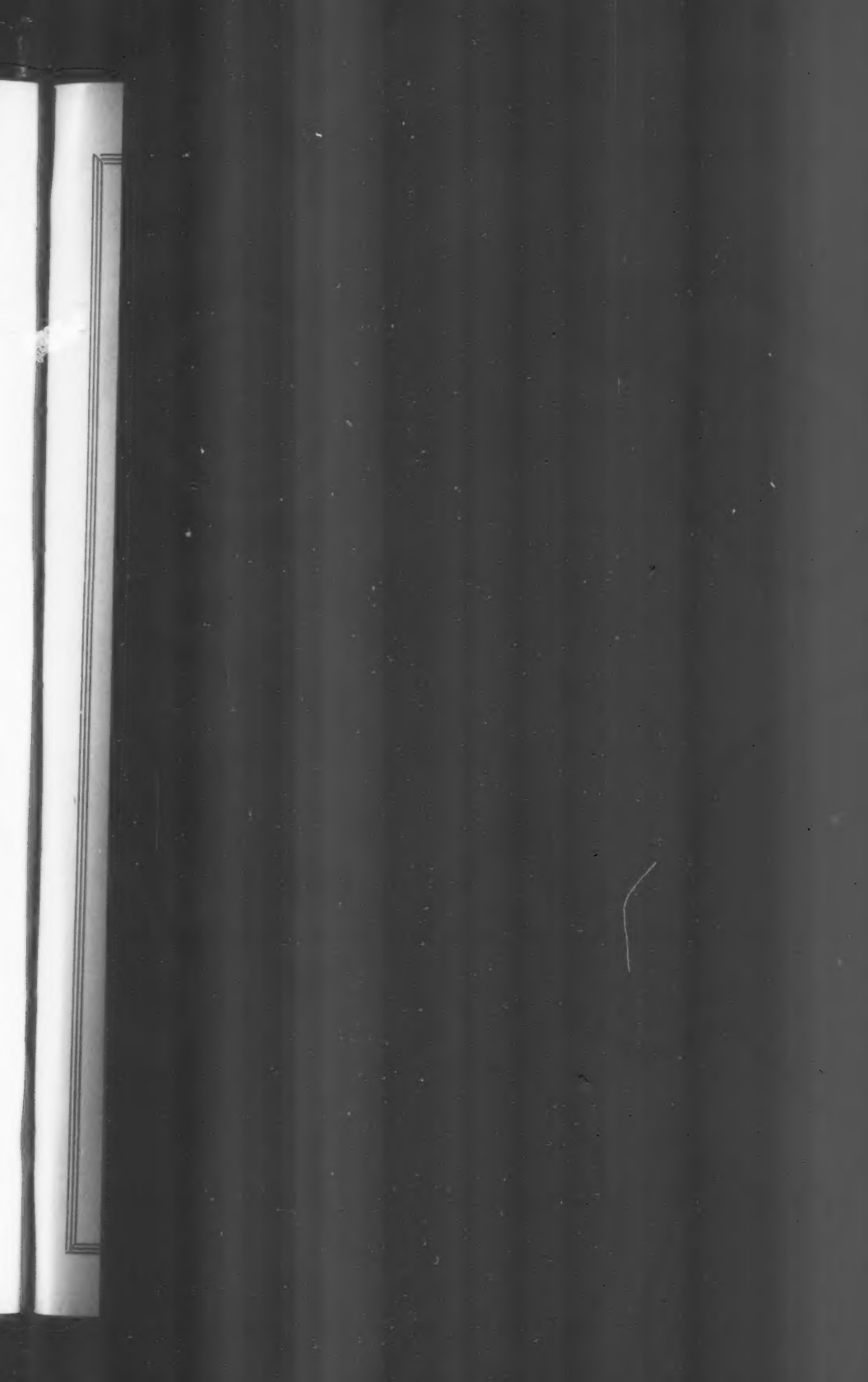

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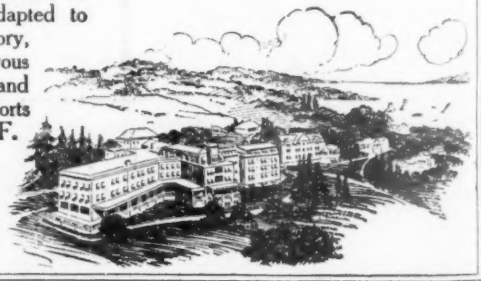

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